VOL. 32 JULY 1956 NO. 7

BULLETIN

OF THE NEW YORK ACADEMY OF MEDICINE

Original Articles by

ROBERT H. KENNEDY

CONDICT W. CUTLER, Jr.

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AGING AND ATHEROSCLEROSIS

Transcription of a Panel Meeting

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Batterman, R. C.; DeGraff, A. C., and Rose, O. A.: Circulation 5:201 (Feb.) 1952.

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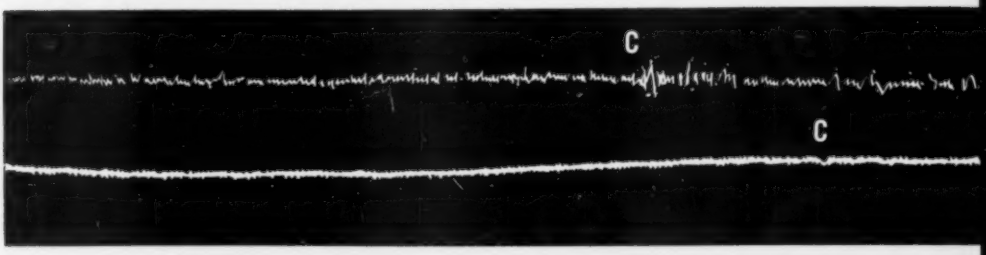

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[1]Albertson, H. A. and Trout, H. H., Jr.; *Antibiotics Annual* 1954-55, Medical Encyclopedia, Inc., New York, N. Y., 1955, pp. 599-602.

[2]Prigot, A.; Whitaker, J. C.; Shidlovsky, B. A., and Marmell, M.: *ibid*, pp. 603-607.

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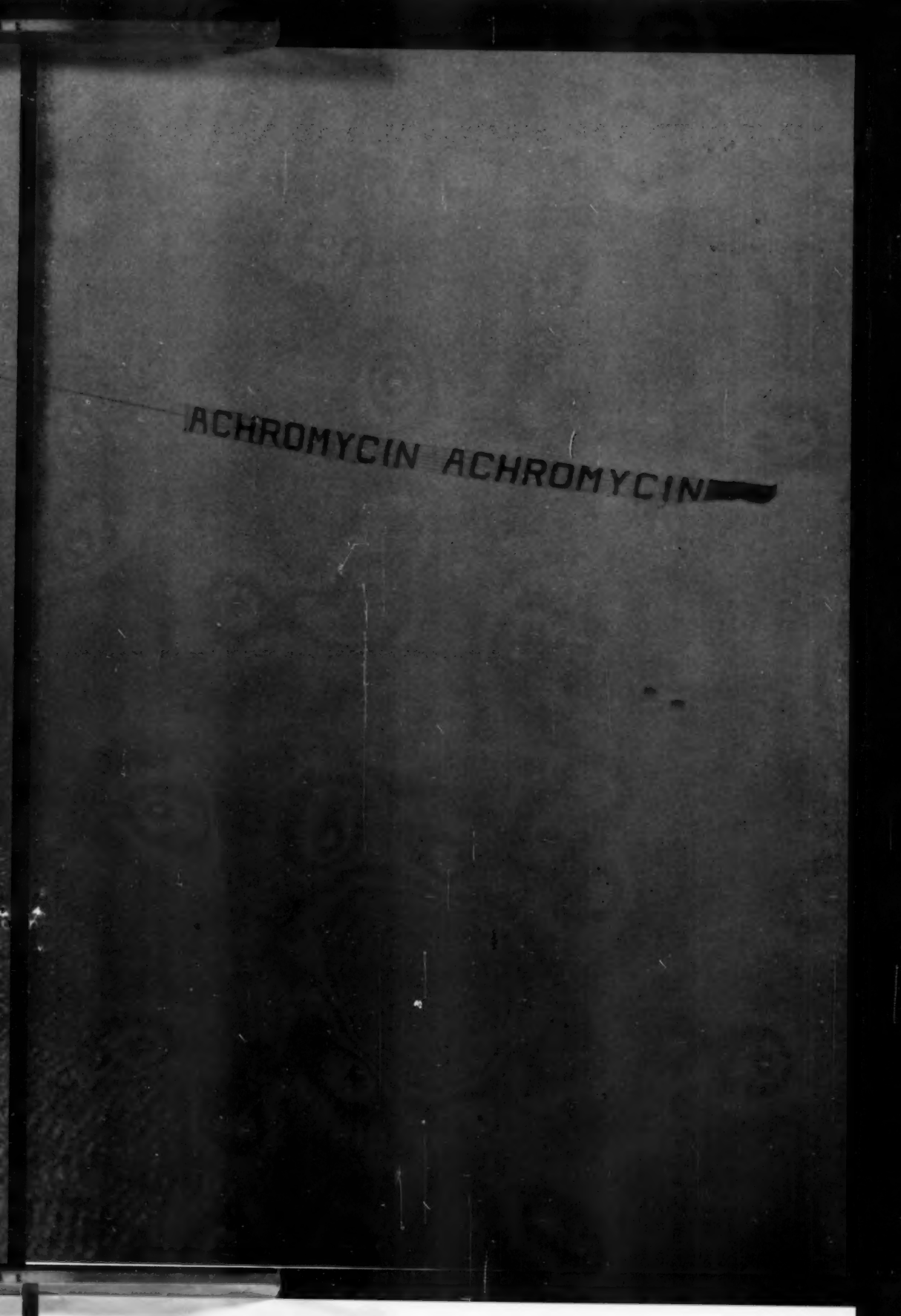
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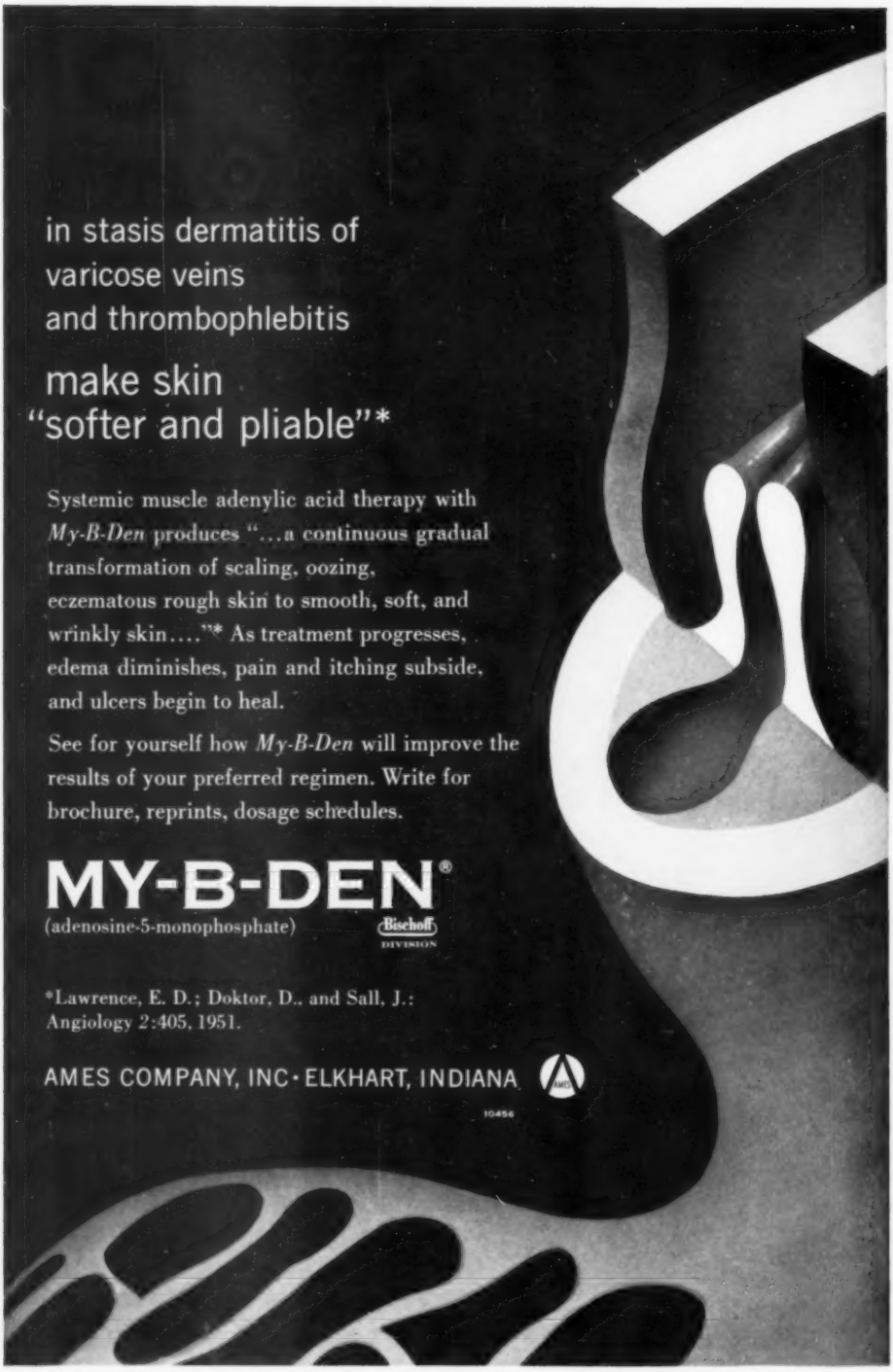

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*Lawrence, E. D.; Doktor, D., and Sall, J.: Angiology 2:405, 1951.

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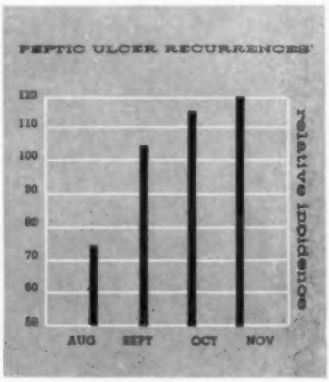

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(1) Ivy, A. C.; Grossman, M. I., and Bachrach, W. H.: Peptic Ulcer, Philadelphia, The Blakiston Company, 1950, p. 626. (2) Steigmann, F. and Dolehide, R. A.: Am. J. Digest. Dis. *22*:37, 1955. (3) Riese, J. A.: Am. J. Gastroenterol. *23*:223, 1955.

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1. Davidson, D. T., Jr.; Lombroso, C., & Markham, C. H.: *New England J. Med.* 253:173, 1955.
2. Zimmerman, F. T.: *New York J. Med.* 55:2338, 1955.

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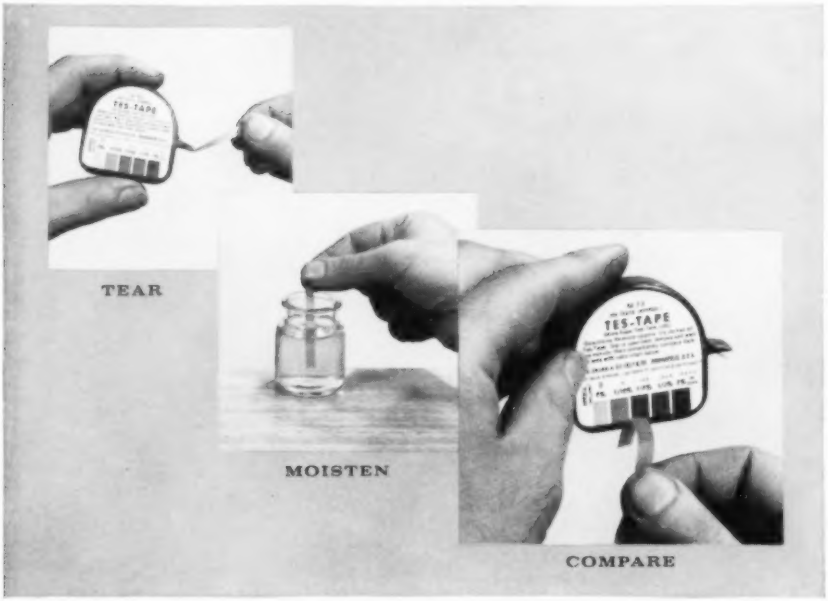

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ROBERT L. CRAIG, *Editor*

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BULLETIN OF
THE NEW YORK ACADEMY
OF MEDICINE

VOL. 32, NO. 7 JULY 1956

MANAGEMENT OF INJURIES IN THE AGED*

ROBERT H. KENNEDY

President, American Association for the Surgery of Trauma; Surgical Director, Beekman-Downtown Hospital, New York, N. Y.

It is estimated that almost ten million accidental injuries occur in this country each year. I have found no statement of the percentage of these injuries in persons over 65 years of age. Among causes of death, however, accidents in this group are responsible for 213 in each 100,000 persons in this age group, following immediately in frequency cardiovascular diseases and cancer. Therefore the number injured must be great and for the foreseeable future the management of accidents in the aged will be a large problem for all of us.

The actual definitive treatment of the injury itself varies little in the advanced years from that in younger persons. The whole problem of management of the aged individual who has suffered an injury includes many important, and even critical, factors not usually stressed in younger persons.

General Considerations:—The extent of activity of the person prior

* Presented before the 28th Graduate Fortnight on *Problems of Aging*, at The New York Academy of Medicine, Ocober 18, 1955.

to his accident needs consideration. The majority were up and about doing something of importance to them or they would not have been in a situation where they could sustain an accident. Our aim must be to return them to a condition where they can accomplish as much as before the accident in the shortest possible time and with the least risk. The minority were mentally incompetent, bedridden or chair-fast before the accident. They may have sustained even a spontaneous fracture from osteoporosis, or a pathologic fracture from chronic bone disease. It is not rational to subject them to some treatment which requires early ambulation, either with or without crutches, when they have not walked for months or years. They should have immediate immobilization in such a manner, however, that nursing care is practical and painless and that deterioration will not result from being allowed to lie like a log. We must analyze the human material which was present before the accident.

Whether accustomed to physical activity or not, were they mentally and emotionally interested before the accident? How did they spend their time? Did they have jobs? If so, is the nature of the injury likely to prevent them from returning to the same work? Can they do any part of their regular work while convalescing?

Has some hobby kept them alert and stimulated to go on living? May the injury keep them permanently from continuing to pursue this hobby? Can it be kept up equally well during the period of bed and chair life required for recovery?

In spite of age, has there been a certain earning capacity and will economic independence now be lost? Was there just enough assured income to pay expenses and no extra cushion to settle bills resulting from hospitalization of unknown duration? Is the psychological reaction to prefer death rather than to become a burden on anyone,—family, friends, or society?

Often confinement following injury is the first time in years that a person has stopped to survey the usefulness and future of his life and he feels that he has come to a "dead end." Strictly he is no different immediately after his injury than before except for one or more broken parts, which will usually mend in time. He is no sicker than previous to the accident and he must not be allowed to feel that he is. He should not be pampered by family or friends or have to listen to everlasting inquiries as to how he feels. He should be treated as a well person and urged to

carry on every activity possible within the limits of damage to his broken part.

The attending physician must be aware of and advisor in all these factors. They are at least equally as important as the care of the injured part. A well healed fracture is no asset in a dead person or in one whose major ambition is to die. Exercise of all uninjured parts should be insisted on from the first day. This does not mean baking, massage and diathermy. It means doctor's instructions for active motion of every joint not immediately adjacent to the injured part several times a day, at definite times and for definite periods. We are not trying to make an athlete out of this elderly person, but to keep him at least as active as before. Since he will not be able to rehabilitate himself as regards joints and muscles as readily as a younger person, why allow him to deteriorate in the first place? This concerns head, neck, muscles of respiration, abdominal muscles, back and all four extremities, with as much attention to finger joints as to those of the shoulder and wrist. If a person will need crutches to walk with, his triceps muscles will bear a major part of the burden. Lifting his trunk by means of his hands on an overhead bar exercises biceps only and will not help him for future crutch walking. With his hands placed on the bed beside and behind his trunk he needs to lift himself many times a day. This will develop his triceps and make crutch walking possible when he is ready for it. Also, with crutch walking he will probably bear weight on one foot only. To be able to bear 100 per cent of his weight on one foot requires that he actively exercise his good leg regularly and effectively from the day of injury so that it can play its part promptly in his recovery at the proper time.

In addition to insisting on physical activity, the physician must constantly urge from the day of injury continuation of work, social activities or hobbies or encourage introduction to new work or new hobbies which will really absorb the individual's interest and fill his time. If there is an economic problem or source of worry, it is the physician's duty to explore this area and propose possible plans for the future as far as possible in the first few days, otherwise his patient may not recover. If there will be permanent crippling or incapacity this situation must be faced in the early days and possible solutions made known to the patient. There is no value in postponing thorough discussion. Probably he knows what he is up against fully as well as the physician does and

indecision as to the future will delay his recovery much more than to be given a possible definite solution for him to work toward. If he has a grain of humor foster this to the limit.

Habits:—We must pay attention to what these patients ask for. They have probably learned the easiest way for them to live comfortably, otherwise they would not be alive so long. If a man wants to keep on his red flannel undershirt in bed in the summer heat insist that he do so, even if the nurse does try to sneak it away from him. If he has smoked most of his life, let him continue to do so. It is not your duty to try to change life-long habits unless you know that they are harming him. At the same time try to keep him on no more than his usual amount of tobacco since everyone is likely to smoke more when his time is unoccupied. If he is accustomed to alcohol let him exercise his usual tolerance. Persuade the dietitian to cater to his idiosyncrasies in food, within the limits of hospital diets. It may be childish for him not to like spinach, but why try to change him now. No adequate diet will affect the healing of a fracture in the slightest although one would like to see him get the calcium contained in one glass of milk a day unless he is allergic to it. If he is always constipated and takes some medication daily, give him the same thing, even if it is not in the hospital formulary. If he is not constipated usually, do not make him so by continually cleaning out his bowels. If he regularly takes something for sleep, do not try to break him of the habit or to change him to a new drug. In other words, try, as nearly as possible, to let him live the kind of life he has been accustomed to living for so many years.

Importance of Common Symptoms and Complications:—Edema requires even more attention than in younger persons. Its persistence is more likely to result in joint, tendon and muscle adhesions. Elevate early and persistently for this. But remember, particularly in the lower extremities, that elevation even to the horizontal in an arteriosclerotic may result in lack of arterial pulse, meaning anoxemia. Phlebitis is a more common complication at this age. Change in the walls of the veins is likely to preexist; lack of subcutaneous fat and sluggishness in movement may result in greater pressure on vessels with less filling; viscosity may be high due to low fluid intake. Phlebitis needs to be kept in mind continually. Decubitus is too common. I cannot subscribe to the notion that its incidence and treatment are the nurse's responsibility. Certainly good nursing does much to prevent its occurrence, but there is no ex-

cuse for the doctor not knowing that redness of the skin is present long before the actual embolic process appears, and advising and supporting the nurse in routine care.

Prostatic congestion and hypertrophy will be common in the old men. Frequently one learns that there has never been a suggestion of urinary retention before the patient has been required to lie in bed. Catheterization may increase the congestion. Many will require a prostatectomy as soon as the condition of the injured part allows required movement for positioning on the operating table. The ability to void spontaneously rarely seems to return after being upset in these injury cases. I believe that diabetes and arteriosclerosis make little difference in fracture healing, except for the danger of infection and gangrene in open wounds or secondary infections about the feet.

In any type of injury to an arthritic, the process in the joints is likely to be activated with increased pain and disability. Recovery is more prolonged and it is often difficult to determine when symptoms are no longer due to injury, but to activation or increase of the arthritic process. In a back injury the x-ray film may show no abnormality except a minimal arthritis. The pain may be severe and persistent. The ligaments and joint capsule are probably much more thickened than is suggested by the amount of calcium deposit visualized on x-ray. Flexibility is diminished and a minor strain of the back may result in tearing fibers with further pain, scarring and prolonged disability. We cannot dismiss these persons as malingerers or neurotics.

General Care of the Injured:—First care of the injured at the scene of accident, in office or emergency room is the same at all ages, — 1) make certain of an open airway; 2) stop hemorrhage; 3) treat shock; 4) immobilize fractures, preferably by the application of traction in lower extremity injuries.

Morphine is for the relief of pain and in these older people is contraindicated except when they are in pain. Otherwise, on recovering from shock and with reestablishment of circulation, mobilization of the morphine may result in respiratory depression and death. Intravenous fluids, particularly blood, are needed in traumatic shock. In advanced years, these fluids must be administered carefully and no more rapidly than required, since the right side of the heart must be protected early and the kidneys later. Always keep in mind the increased danger of a cerebrovascular accident at the time a high blood pressure returns after

it has been radically lowered by shock. In the elderly there is greater danger of permanent brain, heart or kidney damage from allowing shock and anoxemia to persist, than in younger persons. These oldsters have less reserve to combat prolonged anoxemia. There is often much delay in obtaining medical clearance before a required operative procedure. This is particularly true in fractures of the neck of the femur. Except for shock, the patient will never be in better condition than he is immediately after injury. If he has previously been active he needs no more thorough check-up preoperatively than does a younger person. If he needs operation and is out of shock, waiting for the report on his blood urea nitrogen or blood sugar is not going to be the factor which saves his life. If an abdominal injury is believed to require operation, this should be done as soon as shock has been overcome, the same as in a younger person, without delaying for laboratory reports and arguments as to condition of vital organs,—otherwise death will result.

Factors in Specific Injuries:—When found unconscious, the question frequently arises whether the cerebral accident present has been spontaneous or the result of external trauma. Even with an x-ray film showing a fracture the diagnosis is not proved. The patient may have fallen because of a "stroke" and sustained a fractured skull in the fall. His present unconsciousness may be based on spontaneous cerebral hemorrhage or on extradural or subdural clot or intracerebral damage from the fall. Unless this is kept constantly in mind, tragedy may result from failure to recognize an extradural or subdural clot which might be relieved by craniotomy. Further, negative x-ray findings do not necessarily rule out a contrecoup injury which may be fatal if uncared for. On recovery of consciousness, even with paralysis present, remember that present day treatment calls for early mobilization of the patient, no matter what the primary cause of unconsciousness was.

Do not try to immobilize the chest for fractured ribs by strapping with adhesive plaster or applying a tight binder. This, if done effectively, cuts down the air interchange markedly. The patient needs all the oxygen he can get and needs to be able to cough in case there has been lung injury with foreign material in the bronchi. He does not breathe deeply and tries to restrain a cough on account of pain. Procaine injection of the region of the intercostal nerves adjacent to all injured ribs will relieve pain, and allow deep respiration and cough. It may be repeated two or three times a day, if required. The patient with frac-

tured ribs should not be allowed to endure pain of any great degree.

Fractures of the surgical neck of the humerus are common in the elderly and frequently much over treated. Except when accompanied by dislocation of the head of the humerus or when the shaft rides by into the axilla, the anatomical position is usually of little moment and should be practically disregarded. Function of the shoulder muscles needs to be preserved at all costs, since only with these muscles working can we place the hand where we desire, the main function of the upper extremity. If action of the shoulder is once lost it is unlikely, in the aged, that it will return completely. A sling with the elbow at right angle and one turn of a broad bandage about trunk and arm to hold the arm to the side is all that is required. This is merely to relieve discomfort from soft part damage and prevent its increase. Three to five days is usually the upper limit for retention of the body swathe. Then pendulum exercises at the shoulder are started with the trunk flexed as near to a right angle as possible. These exercises should be carried out for at least six periods during the day.

I have often heard it said that it is not worth while to try to reduce an impacted Colles' fracture in the aged. This is heresy. There is no valid reason for allowing pain, disability or deformity to persist without trying to return the fragments to normal position. In the first 12 to 24 hours local injection of procaine into the fracture site usually gives good muscle relaxation for reduction. This reduction should be tried once only and not repeated if unsuccessful. The bones are frequently so brittle that repeated attempts at reduction will result in greater comminution. If the position is not changed in attempting reduction, no splints should be applied, but active motion of fingers, wrist and forearm urged from the start. If the impaction is broken up, the forearm and proximal hand should be immobilized in flexion and ulnar adduction for eight weeks. Meantime motion of all finger joints, elbow and shoulder must be carried out to the limit many times a day.

Fractures of the spine without cord signs should not be treated in hyperextension. In fact they should be immobilized in bed for the briefest time possible to relieve the major soft part pain. The spinal musculature must be kept active if it is expected to continue to support these elderly people for the remainder of their lives.

One of the great advances in surgery in my lifetime has been the general adoption of nailing for fractures of the neck of the femur and

the intertrochanteric region. Previously, the defeatist attitude in the care of these fractures resulted in a tremendous mortality and commonly a lifetime morbidity if they survived. Internal fixation makes these people comfortable, allows competent nursing care and makes bed rest unnecessary if the general condition should require that the patient be up. If displaced, an attempt at anatomic reduction should be made in all cases before the nail is inserted. Unless satisfactory reduction is obtained union cannot be expected. The procedure is not shocking. The sicker the patient the more the procedure is indicated.

In complicated open fractures, early amputation may often be preferable to prolonged care in the presence of sepsis. Attempts to preserve a part by prolonged hospital care may be worth while in a younger person, but foolhardy in the aged who have not too long a future.

Pathological fractures, such as through an area of metastatic carcinoma in bone, deserve just as adequate fracture treatment as any other, since the patient can thereby be made comfortable and the fracture will frequently unite.

We must manage our aged who suffer injury in a way to assure as few useless cripples as possible. For the individual and for society, mental, emotional, or economic crippling following accident is just as much a loss as physical disability. We must treat the injured part to the best of our knowledge and at the same time instill in the individual hope for the future.

MANAGEMENT OF SURGICAL EMERGENCIES IN THE AGED*

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WHEN an acute illness requiring surgery attacks a person deteriorated by age, who is, perhaps, the victim of chronic disease as well, the effect may be catastrophic but it is not necessarily disastrous. To avert disaster the physician must be alert to recognize the situation and prompt in his decision. He must persuade an unwilling family and a reluctant patient to accept surgical aid. To do this conscientiously he must be convinced that this course presents the best, if not the only, hope for the patient and that the chances of recovery are good enough to warrant it.

The purpose of this paper is to examine the common surgical emergencies which beset elderly people and to evaluate the results of surgery in dealing with them. The more frequent emergencies are gangrene of the extremities, diseases of the biliary tract, intestinal obstruction, perforation and hemorrhage from peptic ulceration, mesenteric thrombosis and appendicitis.

Complications: A study of 204 consecutive emergencies in patients averaging 74 years of age gives a fair sample of complicating abnormalities. (Fig. 1.) Ninety per cent of them showed more or less advanced arteriosclerosis, while 67 per cent had clinical evidence of arteriosclerotic heart disease. Twenty-five per cent had nephritis, 18 per cent diabetes, 13 per cent had had coronary occlusions, 12 per cent cerebral vascular accidents. Eight per cent suffered from chronic anemia of various types. Eight per cent had central nervous system disturbance (such as lues or parkinsonism), and another 8 per cent presented prostatism. Eight and a half per cent had decubitus ulcers, indicative of the fact that many were already bedridden. Another 22 per cent showed a variety of chronic ailments including lues, tuberculosis, cancer, unhealed fractures

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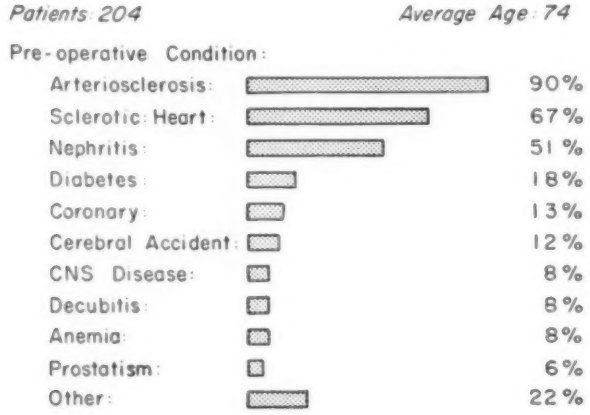

Fig. 1—Incidence of complicating conditions in patients presenting surgical emergencies.

of the femur, amputated legs and various deformities. Untabulated, though frequently encountered, were obesity, malnutrition, vitamin deficiency states, blood volume deficit and psychic disturbance. Since these patients were indigent inmates of city institutions, the incidence of complicating disease may have been higher than would commonly be seen in private practice. They presented a severe test of the results of surgery under adverse circumstances. It was possible to save more than half of them.

General Principles: In dealing with such patients certain general principles were learned:

1. Early recognition of the emergency and prompt intervention to relieve it.

   Older patients succumb very rapidly to the effects of hemorrhage, shock, dehydration, sepsis and pain. Preparation for operation must be limited to restoring lost fluids, electrolytes and blood, correcting acidosis, giving support to the heart and attempting to control sepsis with antibiotics. Too prolonged an effort to achieve an optimal condition for operation may deny the patient his chance for life.
2. Restricting the operation to the least and simplest procedure required for the control of the emergency.

Curative and reconstructive operations are best deferred until better condition of the patient is attained and better preparation is possible.

3. Providing maximum protection of the patient during operation. This involves the correct choice and skilful employment of the anesthetic. We have ceased to regard anesthesia as a major hazard. In 434 bad risk cases only one death was attributable to anesthesia Blood is necessary for support and replacement during operation, and keeping the loss of blood to a minimum is also of great importance. Exact and delicate handling of tissues and the avoidance of rough manipulation are necessary. Gentleness is more important than speed.
4. Using every means to promote the patient's recovery and to prevent complications.

A brief survey of the commoner emergencies will serve to illustrate the application of these principles.

Gangrene of the Extremities: In our group of 204 patients there were 73 cases of gangrene. All were associated with arteriosclerosis. Twenty-nine were diabetic. Forty-five showed sepsis. We were not here concerned with gradually encroaching occlusion of the arterial flow where sympathectomy might give temporary relief or where resection and replacement of partially occluded major vessels might be in order. Nor yet were we dealing with the gradual mummification of one or more toes where elective transmetatarsal amputation or demarcation and separation under conservative management might suffice. We were faced here by the abrupt serious compromise of the circulation by embolism, arteriosclerotic thrombus or the fulminating spread of septic gangrene. In such circumstances the sooner a thigh amputation could be done the better. Temporizing measures such as local amputation of necrotic toes, excision of gangrenous areas or incision for spreading infection of the foot were often followed by rapid spread of the disease necessitating amputation two or three days later. Amputations below the knee gave unsatisfactory results.

Suture of the stump, in septic gangrene, resulted too often in a breaking down of the wound. The stumps were healthier, healed better and the incidence of secondary venous thrombosis was less when the amputation was done without a tourniquet, bleeding being controlled

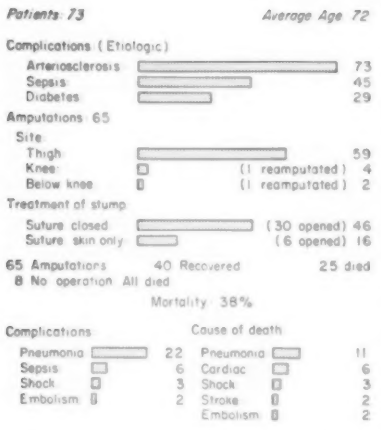

Fig. 2—Causes, treatment and results in gangrene of the extremities.

Fig. 3—Pathology, operation and results in acute biliary tract disease.

by accurate isolation, clamping and ligation of vessels. The tourniquet proved useful in fulminating sepsis with diabetic gangrene where segregation of the leg with refrigeration greatly diminished septicemia, lowered the fever and permitted time for control of acidosis, transfusion and other preparatory measures. As a means of anesthesia we soon abandoned the crymal method, as we did also block and spinal anesthesias. Experience with various methods led us to accept general anesthesia as the safest and most comfortable method for this operation.

The mortality for this emergency, including eight patients who refused operation and died, was 38 per cent. (Fig. 2.)

Biliary Tract Disease: Forty-three biliary tract cases in patients averaging 73 years of age occurred in our group of 204 consecutive emergencies. Gangrene and perforation of the gallbladder occurred early in cholecystitis. Of the 28 cases of cholecystitis, eight gallbladders were found to be gangrenous at operation 48 hours after the onset of symptoms. Four of these already had peritonitis, of which they died. Many were not seen until the third or fourth day of the disease. Occlusions of the common duct were still later in obtaining surgical relief, usually about ten days, and were deeply jaundiced. This delay was due to the difficulty of accurate diagnosis.

We were at first over-persuaded that the one proper operation for

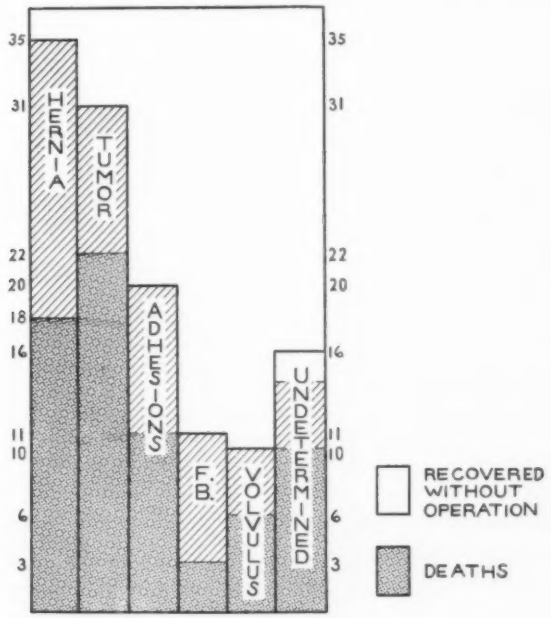

Fig. 4—Causes and results of acute intestinal obstruction.*

cholecystitis was cholecystectomy. Under this policy our mortality approached 60 per cent. Although cholecystostomy was reserved for the most seriously ill, the mortality fell to 35 per cent when this operation was employed more liberally. Since common duct exploration carried a 73 per cent mortality, cholecystostomy was even employed as an emergency measure in common duct occlusion when the cystic duct was patent. The temporary biliary fistula provided decompression of the liver and control of jaundice until the patient might be better prepared to withstand curative surgery. Of the cases of acute cholecystitis treated by cholecystostomy, only 30 per cent required subsequent surgery which, performed as elective operations, the patients could be brought into much better condition to withstand. (Fig. 3.)

This by no means implies that no patient over 60 should have cholecystectomy performed for acute cholecystitis. It is still the opera-

* Figures 4-12 inclusive, are reproduced from an article by Cutler, C. W., Jr. Acute intestinal obstruction in the elderly, in *Surgery, Gynecology and Obstetrics*, 94:481-90, April 1952, by permission of *Surgery, Gynecology and Obstetrics*.

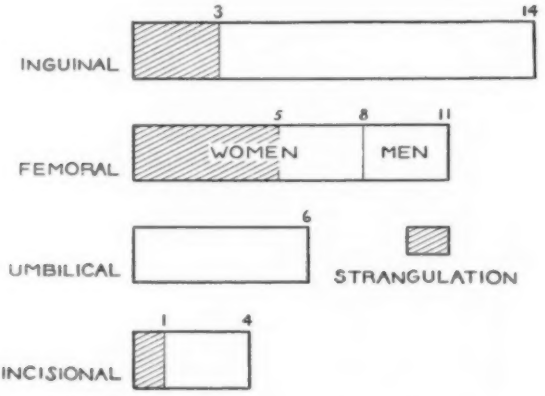

Fig. 5—Hernia as cause of obstruction and relative incidence of strangulation.

tion of choice. For the old patient in poor condition, cholecystostomy is a defensible and respectable operation and may be life-saving.

The mortality in acute biliary tract disease was 49 per cent.

Acute Intestinal Obstruction: The original 204 surgical emergencies in old people produced 56 cases of intestinal obstruction with a mortality of 55 per cent. To this study were added 63 additional cases seen at another hospital. These patients, while of comparable age (average 73 years), were in far better general condition than the first group. Many of them were private patients. In this group the mortality was 53 per per cent. It was apparent from a study of 123 cases that pre-existing disease was not the major factor in determining mortality in obstruction, but rather how and particularly when the patients were treated and the nature and cause of the obstruction. (Fig. 4.)

Obstruction by hernia led the list with 35 cases, next came tumor with 31, then adhesions with 20, foreign bodies with 11, volvulus with ten, and a group of undetermined cause numbering 16.

Among the hernias, the inguinal type accounted for 14 cases, femoral (with a predominance of women victims and an almost 50 per cent incidence of strangulation) produced 11 obstructions, umbilical six and incisional four. (Fig. 5.)

In obstruction from hernia, sudden incarceration and pain with rapid onset of vomiting and both x-ray and clinical evidence of small

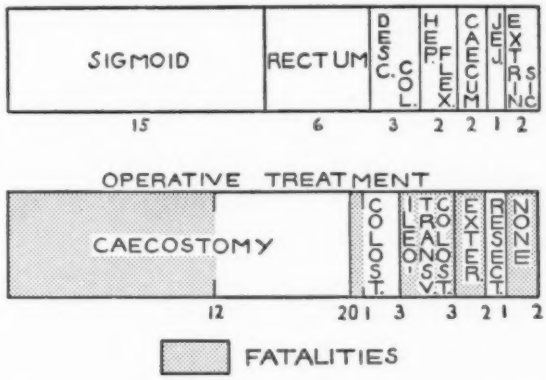

Fig. 6—Tumors causing acute intestinal obstruction. Treatment and results.

bowel distention usually made the diagnosis clear. One femoral hernia, however, escaped detection and another had been reduced with its sac, en masse, both with fatal termination. Four (all femoral) required resection of gangrenous intestine and two survived.

Counting late mortality from apparently unrelated causes and two moribund patients not submitted to surgery, the death rate was 51 per cent. Related postoperative mortality was 33 per cent. These deaths occurred from: pneumonia, four; shock, two; peritonitis, two; congestive heart failure, two; and sepsis, one.

Thirty-one tumors causing obstruction were mostly of the large bowel and generally cancer. Here the onset was often insidious, merging with habitual constipation and delaying recognition for from two to ten days. X-ray barium enema studies provided confirmation of the diagnosis. Cecostomy or divided colostomy was the treatment generally selected and 12 of 20 patients died. Attempts at more formidable procedures met with failure. Two exteriorization resections (Mikulicz) and three ileocolostomies were all attended by fatality. The death rate in the entire tumor group, including late deaths and two patients not operated, was 71 per cent. Related postoperative mortality was 62 per cent. The early causes of death were: peritonitis, six; pneumonia, five; shock, four; cardiac failure, two; uremia, one. The depleting effects of cancer and the delay in diagnosis were held responsible for the high death rate. (Fig. 6.)

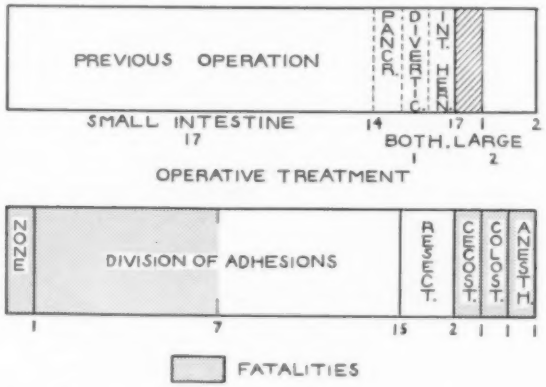

Fig. 7—Adhesions causing obstruction. Etiology, operations and results.

Peritoneal adhesions were responsible for 20 obstructions. All but three had undergone earlier operations which helped in establishing the diagnosis. The remainder had histories suggestive of previous pancreatitis, appendicitis and diverticulitis. In 17 cases the small bowel alone was involved, in two the large bowel and in one, both. Sudden onset of pain with severe cramps and early and persistent vomiting usually characterized these cases and x-ray showed small bowel distention. Relief of the obstruction by simple division of adhesions was possible in 15 instances. Two patients requiring resection following exteriorization of a loop of ileum, survived. Two others in which surgery of the large bowel and ovary, respectively, were added did not. The related postoperative mortality was 40 per cent. Adding two late deaths and one unoperated case raised this rate to 55 per cent. (Fig. 7.)

Foreign bodies caused obstruction in 11 cases. Eight were caused by gallstones and three from ingested foreign bodies—one of them a Miller-Abbott tube. They presented an abrupt and dramatic onset. X-ray usually failed to show the foreign body. The history of earlier attacks of cholecystitis was in some instances suggestive. Where dislodging the offending object could be done without opening the bowel the results were good. Enterotomy increased the hazard, two of six being lost. The overall mortality was 27 per cent, including one unoperated moribund patient. (Fig. 8.)

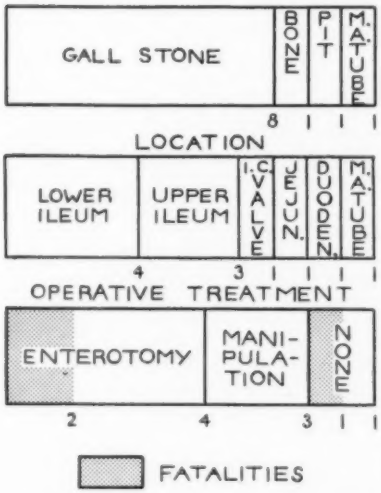

Fig. 8—Nature and location of obstructing foreign bodies. Treatment and results.

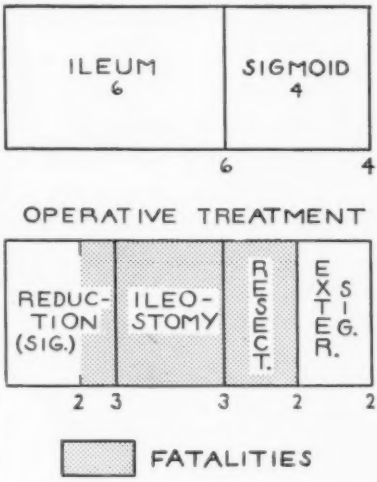

Fig. 9—Location and results of treatment in obstructions due to volvulus.

There were ten cases of acute volvulus. The onset was abrupt and severe as a rule, with sharp, crampy pain. The x-ray was helpful in detecting volvulus of the large bowel. In the small bowel it failed to indicate the etiology. Early gangrene required resection, ileostomy or exteriorization in seven cases. Two patients who had exteriorization of a large bowel loop survived, together with three in whom simple manipulation sufficed. The mortality was 50 per cent. (Fig. 9.)

There were probably other undiagnosed cases of volvulus occurring in a group of unclassified obstructions, 16 in number. In four of these, operation failed to disclose the source of trouble. Four were relieved by non-surgical treatment, while eight died without operation or subsequent autopsy being performed. This group had a 62 per cent mortality. (Fig. 10.)

There was a distinct relationship between the rate of survival in obstruction cases and the rapidity with which surgical relief was effected. Simplicity of procedure also had a favorable effect. Prolonged attempts at treatment by irrigations and intubation were a distinctly unfavorable factor. The mortality among patients operated upon within 24 hours was 39 per cent as contrasted with 60 per cent in those treated

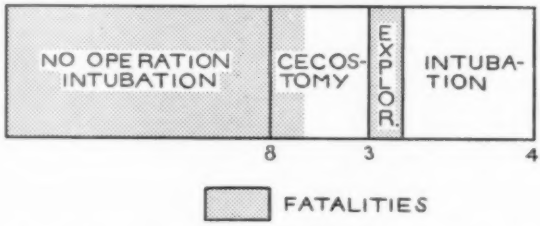

Fig. 10—Obstructions of undetermined cause and results of treatment.

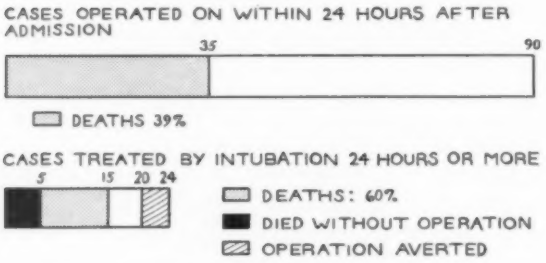

Fig. 11—Results of intubation in the treatment of acute obstruction.

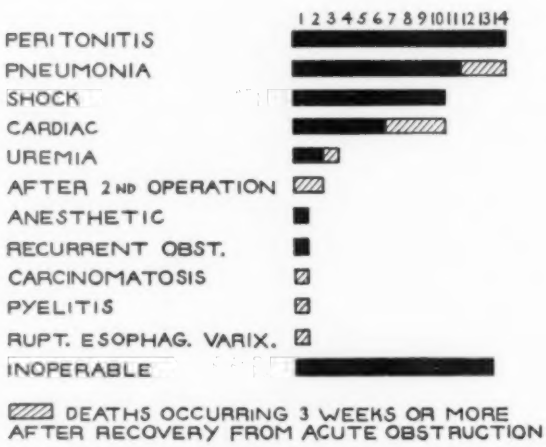

Fig. 12—Causes of death in acute intestinal obstruction.

by intubation for 24 hours or more. In only four instances in 30 cases did protracted intubation avert operation, while five patients thus treated died without operation. (Fig. 11.)

In the obstructed patients peritonitis, pneumonia and shock accounted for most of the early postoperative deaths (47 per cent). Late deaths and unoperated cases, 97 per cent fatal, raised the rate to 57 per cent. (Fig. 12.)

Complications of Peptic Ulcer: The records of a ten-year period at a general hospital reveal a total of 40 cases of massive hemorrhage from the upper gastrointestinal tract in patients averaging 69 years of age. Eighteen of these were from gastric ulcer, two from cancer and 20 were from duodenal ulcers.

Many had long-standing complaints suggesting ulcer. X-ray study often proved helpful in localizing the ulcer and in ruling out esophageal varices.

When the ulcer was small and difficult to palpate, gastrotomy was performed to locate it and control it by suture. The patient's condition improved rapidly, even on the table, as soon as bleeding was stopped by this means or by ligation of the gastroduodenal artery.

Unless massive hemorrhage came quickly under control, or if it recurred, prompt resection was considered obligatory. While bleeding patients might be kept alive for some time by repeated transfusions, they did not do as well when finally subjected to operation as did those not so largely dependent on borrowed blood. The patients who died were not our oldest patients, nor yet those most afflicted with incidental ailments, but rather those who had bled longest.

In all but one case (pyloric exclusion) subtotal gastrectomy was performed. Deaths were two of 18 resections for stomach ulcer, three of 20 duodenal ulcer. The patient receiving pyloric exclusion died. Both cancer cases survived to leave the hospital. The postoperative mortality was 15 per cent. (Fig. 13.)

Of all ulcer patients over 60, 10 per cent suffered perforation. There were 18 gastric ulcer perforations and 39 duodenal in patients whose average age was 70. Men outnumbered women by 50 to 7.

While the possibility of coronary occlusion or pancreatitis was sometimes considered, the dramatic onset of severe abdominal pain with shock and a rapidly developing board-like rigidity, strongly suggested the diagnosis. X-ray did not always show air beneath the diaphragm.

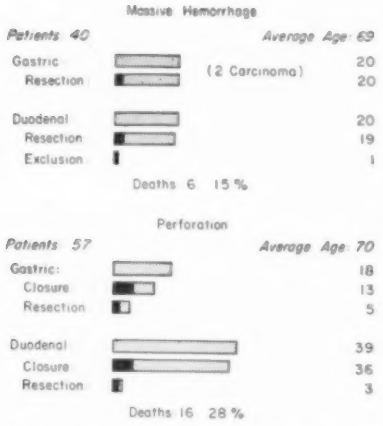

Fig. 13 — Emergency complications of peptic ulcer. Treatment and results.

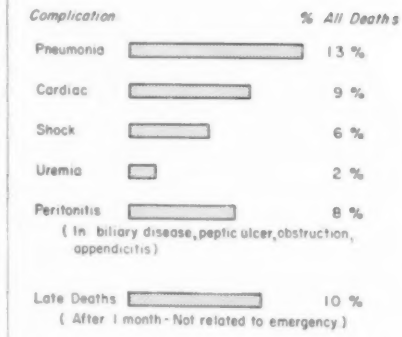

Fig. 14 — Relative incidence of early causes of death following surgical emergencies and of late, unrelated deaths.

Twelve of the 18 gastric perforations were treated by simple closure with six deaths, five by sub-total gastrectomy with two deaths, and one by closure to which gastro-enterostomy was added. Of 39 duodenal perforations, 36 had simple closure and six of these were lost, while subtotal gastrectomy was done in three, with two deaths. The mortality of the gastric cases ran to 44 per cent, while that of the duodenal perforations was 20 per cent. The overall mortality for this emergency was 28 per cent. (Fig. 13.)

Mesenteric Occlusion: Ten cases of mesenteric vascular occlusion in patients over 60 were recorded in a ten year period. The average age was 71. All were victims of advanced arteriosclerosis. Abdominal pain of uncharacteristic distribution and varying severity marked the onset. Tenderness and pain were present. Vomiting occurred in two instances, diarrhea in two. Distention was gradual. Prostration was marked as a rule and was well developed at the time of admission, usually three or four days after the onset. Fever was not pronounced, but a high leukocyte count with polynucleosis was almost invariably present. X-ray did little to establish the diagnoses, which were usually faulty, including intestinal obstruction, gastroenteritis, pneumonia, pancreatitis, perforated peptic ulcer and coronary occlusion. For this reason only four were brought to operation and none under four days after onset.

All of these ten patients died, whether operated upon or not. Possibly earlier recognition and prompt surgery might save an occasional one.

Acute Appendicitis: In the original group of 204 surgical emergencies in elderly patients, appendicitis occurred but five times. The early symptoms were often misleading and the physical signs equivocal. The diagnosis was made eight times. It proved incorrect in four, while one case diagnosed as intestinal obstruction had gangrenous appendicitis with peritonitis. This patient died, as did one of the four correctly diagnosed cases. Three, therefore, were found to have gangrene and perforation of the appendix although their symptoms had been of one day's duration or less. The fourth had a large abscess. The mortality rate was 40 per cent.

In the older age group, as in children, early perforation is to be anticipated, often with minimal early signs. The peritoneum does not readily control or isolate the infection. It is important to be attentive to complaints of abdominal pain in old patients, to be watchful of the temperature, blood count and particularly of the pulse, and to submit the suspects promptly to surgery.

Postoperative Care: Correctness of postoperative care is of particular importance. Oxygen in the early hours is of definite value. Rolling and turning the patient are best postponed until pulse and blood pressure are stable and shock abated. Getting the patient out of bed within 24 or 48 hours has been beneficial.

Adequate administration of fluids and electrolytes is necessary, but with care not to overload a precarious circulatory apparatus with too rapidly administered intravenous liquids or incompetent kidneys with too much salt. The alimentary tract is the best route for supplying fluids, salts, vitamins and nutriment and as soon as possible the patients should be urged and encouraged to take them by mouth. The protein hydrolysates have proved very valuable in oral feeding to restore nitrogen levels. Blood transfusions are often needed.

Older people are peculiarly susceptible to morphine and its derivatives, while many are excited or abnormally depressed by barbiturates. The use of anticoagulants, heparin and dicumarol, requires caution since their effects are highly unpredictable and often, even in small dosage, may cause dangerous prolongation of bleeding, clotting and prothrombin time of uncertain duration and difficult of control. About half of our patients were treated before the days of penicillin. The advent

of organic antibiotics led to the hope that we might control pneumonia, or even prevent it. Actually we found relatively little difference in its incidence or mortality rate under their use. Undesirable by-effects of antibiotics have led us to abandon their use as a routine preventive measure.

Giving hope, encouragement, sympathy, confidence and personal interest to the elderly patient has incalculable therapeutic value.

Morbidity and Mortality: Pneumonia was the greatest killer, with circulatory disturbances a close second. Shock ranked third. In abdominal emergencies, peritonitis was a serious hazard. Renal insufficiency was a mortality factor in all groups. Anesthesia accounted for but one death among 434 cases. Taking all emergencies into account, the mortality rate of operated cases approximated 40 per cent. The rate among unoperated cases ran above 95 per cent. (Fig. 14.)

To the early postoperative mortality directly related to the emergency there was an additional 10 per cent occurring several weeks or months afterward due to pre-existing or incidental ailments. These deaths resulted from pneumonia, coronary occlusion, heart failure, cerebral vascular accidents and the like. Some of these fatalities were probably due to the upsetting of a delicate homeostatic balance. The surgeon should accept this as part of the hazard. Such deaths are included in our mortality figures.

The elderly patient's chances of recovery from a catastrophic surgical emergency today are much better than the figures here cited would indicate. These observations were made for the most part on very bad risk cases at a time when we were just learning to meet the problems of urgent geriatric surgery and before many modern refinements of diagnosis and treatment were available to us, such as accurate determination of blood volume, nitrogen and electrolyte balance, the use of the newer anticoagulants and the organic antibiotics. But even with these handicaps, and accepting responsibility for late deaths not related to the emergency, it was possible to save at least half of these patients by prompt and appropriate operation. Age and decrepitude in the elderly patient afflicted by a life-threatening surgical emergency should not act as a deterrent to the surgeon or as encouragement to procrastination or palliative measures, but rather as a spur and incentive to prompt, effective and simple operative intervention.

INDICATIONS FOR TREATMENT OF PRIMARY TUBERCULOSIS IN CHILDREN*

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UNTIL recently very few physicians were interested in the treatment of children with primary tuberculosis since most children recovered spontaneously without clinical evidence of illness. An unfortunate few developed serious complications such as meningitis and miliary tuberculosis and died since no specific therapy was available. The conquest of tuberculous meningitis has stimulated many physicians to show a new interest in the treatment of primary tuberculosis and its complications. Some physicians advocate only the treatment of patients who are clinically ill; many give specific treatment to children with x-ray evidence of primary tuberculosis regardless of symptoms and still others treat children with a positive tuberculin test as the only evidence of disease.[1] This has led to a confusing situation and all physicians interested in tuberculosis should pause to consider certain facts before deciding on the indications for treatment of the tuberculous child.

In the first place we have as yet no antituberculous agent which will cure the patient of his illness. We have powerful drugs which, if properly used, may achieve a bacteriostasis which will allow the natural defenses of the body to control the tuberculous disease. Tuberculosis is a chronic disease and in most patients recovery will take years in spite of specific therapy.

In the second place we have as yet no good diagnostic test for activity which will help us distinguish between the child whose positive tuberculin test denotes recently acquired and active disease and the child whose tuberculin test converted to positive many years before and whose infection has been arrested for years.

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General Management of the Tuberculous Child: It is important to remember that drug therapy does not eliminate the need for excellent general care and supervision which every child with active tuberculosis should receive regardless of the presence or absence of symptoms. A child with active tuberculosis without symptoms does not need hospital or sanatorium care unless his home is inadequate. Activity should be restricted during the early months of the disease even if the child is afebrile. The child need not be kept in bed but his play must be supervised and playthings selected to minimize activity. Children of school age who are past the acute stage of the disease and are afebrile may attend school if physical activities can be restricted. Diet should be liberal from the standpoint of calories; fat and protein content should be high and supplementary vitamins should be given. The child with active tuberculosis should be protected against excessive amounts of sunlight. If a child with active tuberculosis is exposed to measles, gamma globulin should be given promptly. Vaccination against smallpox should be deferred until the tuberculous disease is inactive except in the event of exposure to smallpox or in the presence of an epidemic. A child with active tuberculosis, even if he is free from symptoms, should be examined by the physician at regular intervals and monthly x-rays are desirable during the active phase of the disease. The necessary duration of conservative therapy will vary with factors such as the extent of the tuberculous disease and the age of the child. Usually increased activity is permitted after the disease is estimated to have existed for six months and in the average case restriction is not continued beyond a year when the major danger of complications is over.

Selection of Cases for Specific Therapy: The selection of cases for whom specific therapy is needed in addition to conservative treatment is determined largely by the estimated prognosis of each individual patient. It is obviously necessary to treat children with forms of tuberculosis which had a high death rate before the advent of specific therapy. Antimicrobial therapy of meningitis and miliary tuberculosis is mandatory. Experience at Bellevue Hospital in the prechemotherapy era disclosed two other main causes of death from primary tuberculosis: 1) locally progressive primary pulmonary tuberculosis with cavitation and bronchogenic spread; and 2) forms of hematogenous tuberculosis more protracted than miliary, in which multiple foci of tuberculosis are found in various parts of the body. These forms of tuberculosis

OUTLINE OF SPECIFIC THERAPY*

CHEST CLINIC OF THE CHILDREN'S MEDICAL SERVICE OF BELLEVUE HOSPITAL

TUBERCULOUS MENINGITIS

Streptomycin:	intramuscular for 6 months	—1.0 gm. daily for minimum period of 1 month or until CSF sugar has been normal for 1 week; thereafter b.i.w.
Isoniazid:	oral for 1 year	—10 mg./kg. daily for 4 to 6 weeks; thereafter 7 mg./kg.
Promizole:	oral for 2 years	—0.25 to 8.0 gm. daily.

MILIARY TUBERCULOSIS

Isoniazid:	oral for 1 year	—10 mg./kg. daily for 4 to 6 weeks; thereafter 7 mg./kg.
Promizole:	oral for 2 years	—0.25 to 8.0 gm. daily.
Streptomycin:	intramuscular for 4 months	—1.0 gm. daily; later b.i.w. In some cases streptomycin is omitted.

In other forms of tuberculosis which require therapy, isoniazid is given daily in doses of 5 mg./kg., with daily PAS or Promizole; or streptomycin is given daily or b.i.w. on weight basis combined with daily PAS or Promizole. Streptomycin and isoniazid are rarely given together, except as a life-saving measure. Therapy is frequently prolonged for a year or more.

Dosage Schedule for Streptomycin (WEIGHT BASIS)

0.3 gm.	—intramuscularly	daily	— under 10 pounds
0.4 gm.	— "	"	— 10 thru 19 pounds
0.5 gm.	— "	"	— 20 thru 39 pounds
0.6 gm.	— "	"	— 40 thru 59 pounds
0.75 gm.	— "	"	— 60 thru 89 pounds
1.0 gm.	— "	"	— 90 pounds and over

Isoniazid:	Given by mouth every 12 hours. In case of vomiting, same amount may be given intramuscularly.
Streptomycin:	A 1.0 gm. daily dose is sometimes divided in two for comfort of the patient. Smaller doses are given as single daily injections except in small or emaciated patients.
Promizole:	Given by mouth every 6 hours. In absence of signs of toxicity a gradual step-like increase from 0.5 gm. is made every few days until a blood level of 1-3 mg.% is obtained (take 2½-3 hours after last dose of Promizole).
PAS:	Para-aminosalicylic acid. Given by mouth every 4 hours for 3 to 4 daily doses, 0.5 gm./kg./24 hours up to maximum of 12 gm. daily dose.

In patients in whom resistance to the major drugs has developed, *Viomycin* and *Pyrazinamide* may be used.

* Subject to modification for individual cases.

must be treated as a life saving measure.

The method of treatment will depend in part on knowledge of the clinical course of the complications when untreated. Many schemes of therapy have been reported and there is certainly no one method that is invariably successful. Details of drugs and schemes of treatment in use in our clinic are shown on the accompanying chart. The essentials for success are early diagnosis, prompt and vigorous treatment and prolonged therapy. Since untreated tuberculous meningitis was practically always fatal and the average duration of this complication was less than three weeks, the two most efficient antimicrobial agents, streptomycin and isoniazid, should be used concurrently and in maximum dosage, lowering the dosage only when the patient seems to be making a clinical recovery. Many physicians treat miliary tuberculosis as intensively as meningitis, using both streptomycin and isoniazid, because of the very high mortality rate in untreated cases. Excellent results however have been reported without the use of streptomycin.[2]

The concurrent use of both the most effective antimicrobial agents is usually reserved for children with meningitis or miliary tuberculosis except in patients with fulminating disease. Since tuberculosis is a chronic disease and arrest, not cure, is the most we can hope to achieve, it would seem wiser to reserve one potent antituberculous agent for possible later use. Because of its minimal toxic side effects and ease of administration isoniazid rather than streptomycin is used in the treatment of protracted hematogenous tuberculosis and locally progressive primary tuberculosis. In addition isoniazid is to be preferred to streptomycin because there is increasing evidence that meningitis and miliary tuberculosis do not develop in children who are receiving adequate treatment with isoniazid. Some investigators advocate the use of isoniazid alone but most physicians prefer to use isoniazid in combined therapy, with a secondary drug such as Promizole or PAS (para-aminosalicylic acid), Promizole being given in the usual dosage estimated by blood levels and PAS in dosage of 0.5 gm/kg. with a maximum daily dose of 12 gm. The purpose of combined therapy is mainly to delay the emergence of resistant tubercle bacilli. Treatment should be continued for at least a year and for three to six months after signs and symptoms have disappeared and the disease is apparently under control. Cavitating primary tuberculosis is considered controlled when cultures from gastric washings have been negative, x-rays stabilized

and cavities closed for a period of six months. If these objectives cannot be reached, resection of the residual disease should be considered while the child is still receiving antimicrobial therapy.

During the years 1930-1946 before streptomycin was available, the mortality rate among the children with primary tuberculosis seen at Bellevue Hospital was 21.5 per cent.[3] With the use of specific therapy deaths have become uncommon and the death rate in 1953-54 was only 1.0 per cent. The aim of therapy however should not only be survival but survival in the best possible condition. In most of the serious complications of primary tuberculosis in which treatment is mandatory, prompt diagnosis and prolonged treatment result not only in a better survival rate but in less residual disease in the survivors.

In addition to the use of specific treatment as a life-saving measure antimicrobial therapy may be useful to alleviate symptoms, to shorten the duration of illness and to minimize the incidence of sequelae. For example, therapy usually has a rapid beneficial effect on tuberculosis of the gastrointestinal and genitourinary tracts, relieving symptoms and often producing roentgen evidence of healing. Many forms of tuberculosis of the skin heal rapidly and draining cutaneous sinuses close promptly, thus permitting surgery of the underlying disease in bones and nodes. The potential danger of dissemination following surgery can be practically eliminated by protecting the tuberculous patient with antimicrobial therapy during a surgical procedure and for a short time thereafter.

In other complications of primary tuberculosis, such as disease of the bones, joints, or lymph nodes, it is difficult to evaluate the result of specific therapy because these complications tend to run a chronic course and may have periods of spontaneous remission even when specific therapy is not given. Specific therapy may be tried in such complications provided only one of the two major drugs is employed and the drug selected is given in minimum effective dosage.

Finally some complications show no visible or apparent response to specific therapy and therefore there is no clear indication for antimicrobial therapy. For example, in endobronchial tuberculosis caused by the encroachment of caseous nodes on bronchi, neither the symptoms nor the appearance of the lesions on bronchoscopy shows a definite response to therapy. In fact, progression of endobronchial disease has been repeatedly observed while the patient is receiving treatment. In

many cases the lesion will undoubtedly heal as well without specific therapy. However in infants with severe symptoms or whenever the occurrence of bronchogenic dissemination seems likely, specific treatment may be given to control the spread of the disease rather than to treat the local condition.

Treatment of Asymptomatic Primary Tuberculosis: So far we have been discussing the treatment of tuberculous children who have clinical evidence of disease. Probably we all agree that treatment of such children is always justifiable and sometimes mandatory. The point on which clinicians disagree is the desirability of treating tuberculous children who are without clinical symptoms or even without roentgen evidence of tuberculosis.

The purpose of treatment is obviously not to alleviate symptoms since these children are free from symptoms. The purpose then can only be to prevent complications especially those which might threaten life. It is well known that streptomycin is not adequate for this purpose, a notable example being the development of meningitis in children under treatment with streptomycin for miliary tuberculosis. There is, however, increasing evidence that clinical meningitis does not develop in children while they are receiving adequate doses of isoniazid. However, before starting a universal program of treatment of all children with primary tuberculosis in order to prevent meningitis it is essential that additional information should be assembled. The most important data to be obtained concern the required duration of therapy. Since 90 per cent of the deaths from primary tuberculosis occur within a year of the discovery of the disease, it would seem reasonable to treat children for a minimum period of a year. Shorter periods of treatment may be inadequate. Information has been obtained regarding the occurrence of meningitis in children who had been treated with isoniazid for only a few months, the first symptom of meningitis appearing one to four months after treatment was discontinued. Another important point to be established is whether isoniazid may be given alone or must be given in combined therapy with another drug in order to prolong its effective use. The optimum dosage of isoniazid must also be established. We have recently seen a patient who developed meningitis after seven months of therapy with isoniazid but who received a maximum of 4 mg./kg. daily. Moreover a treated patient must be followed for a period of years after cessation of therapy in order to learn

whether complications such as meningitis, which usually appear early, have been averted or only postponed in some instances. Finally the frequency of late complications and especially chronic pulmonary tuberculosis during adolescence must be determined in patients who received antimicrobial treatment during early childhood.

A carefully controlled series of children with primary tuberculosis, in which patients are selected at random, must be studied to secure the answers to these important questions. A very large number of cases may be required for this project since the actual incidence of meningitis in primary tuberculosis is unknown.

Since January 1, 1955 a large number of pediatric centers have embarked on a cooperative study of the evaluation of isoniazid in the prevention of meningitis in children with primary tuberculosis. The Public Health Service is coordinating this study which includes infants under the age of three with positive tuberculin tests regardless of the presence or absence of roentgen evidence of tuberculosis and children three years of age and older with x-ray evidence of manifest primary tuberculosis. Children receive either placebo or approximately 5 mg. of isoniazid per kilo and the clinicians do not know which children are receiving the drug. Treatment will be continued for a year and subsequent observations will be continued indefinitely.

Until the results of this study are known it would seem wise to withhold treatment from children with asyptomatic primary tuberculosis who can be kept under close observation. Every physician will want to make an occasional exception when in his judgment it is desirable to treat a particular child.

If the controlled study proves that isoniazid given for a year will prevent meningitis we shall be faced with the task of treating large numbers of children with asymptomatic tuberculosis in order to prevent meningitis in a few. But even if we can prevent meningitis in the child whose tuberculosis has been recognized we shall still be faced with the problem of the child who develops meningitis before his tuberculosis is diagnosed. Any plan for prevention of meningitis must be implemented in the first place by programs for prevention of infection, by case finding among adults and by BCG vaccination when necessary. Secondly we shall need increased case finding among infants and children. Pediatricians must develop a program of tuberculin testing coordinated with established immunization and health examination schedules so that testing

will begin in early infancy and be repeated at regular intervals. If our goal is the elimination of tuberculous meningitis in children we shall need more than just the knowledge that isoniazid prevents meningitis in the tuberculous child.

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AGING AND ATHEROSCLEROSIS

*Transcription of a Panel Meeting**

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MODERATOR SIMMS: This morning's panel discussion is on *Aging and Atherosclerosis*, which represent two extremely important medical problems.

First, I think it might be well to point out that, although this program is unrehearsed, we do have a list of questions so that the other members of the panel will know what to anticipate by way of subjects for discussion.

We have a few general questions. One of these asks for a definition of atherosclerosis. Literally the word means "soupy hardness" but that is hardly a definition of what we mean by atherosclerosis at the present time. Dr. Steele, would you care to venture a definition?

DR. J. MURRAY STEELE: I think to define atherosclerosis is a very difficult task. We are not certain that we are dealing with a single entity but I think Dr. Kendall has probably offered the best definition that there is when we are considering the particular disease that lays down the fatty plaque, the atheroma in the intima. I believe he likes to call it "intimal atherosclerosis" because of the notion that it really begins either in the intima or in the subintima and damage to the remaining part of the artery comes later.

MODERATOR SIMMS: *Dr. Kendall, would you care to comment further on that?*

DR. FORREST E. KENDALL: I was interested to hear that I had a definition! As a biochemist, I feel that I should leave this question of definition to those who have more background in clinical medicine and pathology.

DR. AARON KELLNER: One would think it would be possible at this time to give a fairly precise definition of what we mean by atherosclerosis. Unfortunately, as far as I am concerned, that is not the case. We must still define atherosclerosis essentially on morphological grounds, bearing in mind that what one individual calls atherosclerosis may not necessarily be atherosclerosis to another. My own prejudice as far as the definition is concerned is that atherosclerosis is a disease of the larger arteries which results in thickening of the intima and gradual narrowing of the lumen, and that one of the hallmarks, perhaps *the* hallmark, of the disease is the presence of lipid within the lesion, particularly in the early stages. There are differences of opinion about the role that the lipids play. It is my feeling that the lipids play an essential and fundamental role, that the infiltration of lipids is one of the basic phenomena of atherosclerosis. There are others, Duguid in England for

example, who feel that lipids are secondary, that thrombosis occurring on the intimal surface is the primary event, and the thrombus eventually organizes and produces the narrowing of the lumen. Winternitz in this country had a similar approach to atherosclerosis. He, too, felt that the lipid was secondary, that it resulted from the breakdown of blood clot following hemorrhage into the wall of the vessel. I think, however, that for purposes of this discussion, my colleagues on the panel would probably agree with me that we regard lipids as one of the essential features of the anatomical and perhaps also of the pathogenetic aspects of the disease. I have some photographs of atherosclerosis in human beings that I should like to show as illustrative material, either now or whenever you think it appropriate.

MODERATOR SIMMS: I would like to call on you a little later for that. In that connection it might be pointed out that, whereas atherosclerosis is an extremely important disease in man, it is not important in most animals. We have, as compared with other species, quite a long life span but the early death of most animals does not result from atherosclerosis. Chickens do have it. Cows, dogs, sheep, rats, rabbits don't normally have severe atherosclerosis.

The second question is in regard to the relationship between atherosclerosis and aging. Since this is a Fortnight on aging, it is appropriate that we comment on whether atherosclerosis is part of the aging process or whether, on the other hand, aging influences the incidence of atherosclerosis?

DR. STEELE: I think it has become increasingly clear that atherosclerosis is not necessarily part and parcel of the aging process. It is concomitant with aging. It is seen with increasing frequency with increasing age but one sees persons in the seventh and eighth decades, who die from other ailments, practically without atherosclerosis. Furthermore, atherosclerosis occurs in very young persons. The pathologists at McGill, Peck and others, showed at least one shower of fatty plaques in everyone over seven years of age. I believe that was the age they gave. Therefore it seems not to be directly connected with aging although as we live longer, as in many other diseases, we have a better chance to develop atherosclerosis.

MODERATOR SIMMS: Before going on to a discussion of the pathology, I would ask those in the audience who have questions to please write them out. They will be collected and we shall try to answer them to-

ward the end of the program.

With regard to the pathology of this condition I have two questions which I will ask of Dr. Kellner. The first question is to describe the early, as compared with the later, atherosclerotic lesions,—in other words, changes relative to the age of the lesion.

DR. KELLNER: The early lesions of atherosclerosis that we see with the naked eye or with the microscope result from the infiltrations of lipid material through the presumably intact endothelium into the intima of the blood vessel. There it is quickly taken up by phagocytes so that the first thing one sees is a tiny yellowish pin point or fleck on the vessel wall. Early in life one sees these in the aorta and very commonly on the aortic leaflet of the mitral valve. These small lipid aggregates at this stage are probably reversible. It is quite likely that before any other tissue changes ensue the lipid may disappear and leave little or no trace. However, in most human beings the process is a slowly progressive one, perhaps a cyclical one, and more and more lipid continues to be deposited in the vessel wall. As it does, the small lipid-rich plaques become larger and larger, protrude into the lumen and over a period of months and years, undergo secondary changes. These secondary changes are breakdown of the fat-containing cells with liberation of the fat, fibrosis, hyalinization and calcification. As the plaque gets larger and larger and as the lipid in the central portion of the plaque breaks down, blood vessels grow into the periphery of the plaque. Some of these vessels may rupture, producing hemorrhage into the plaque. As the plaque expands, the lumen becomes compromised; it becomes narrower and narrower and finally may be completely occluded. The lumen may become closed by the plaque itself or by thrombosis or hemorrhage superimposed on the plaque. Though we are all deeply interested in the phenomena that produce disability and death in atherosclerosis, namely, occlusion, thrombosis and hemorrhage into the plaque, I want to emphasize the point that, from a broad point of view, these are secondary phenomena. Important, to be sure, but nevertheless secondary. The underlying process, as I see it, is the infiltration of lipids into the vessel wall. Thrombosis and other disastrous accidents that occur are but the sequelae to this process.

(Slide) These few slides represent atherosclerosis as seen in a large metropolitan hospital. This is a very early atherosclerotic plaque just beneath the endothelium. This is intact endothelium, this the intima, and

here a small cluster of pale cells. These are histiocytes filled with lipid.

(Slide) This is a slightly later stage of the same process. Here is the normal endothelium; here you see a small hillock composed essentially of large pale cells which are filled with lipid material, and you see that at this stage it is already beginning to encroach ever so slightly upon the lumen. At this stage and in the preceding stage I think the process is probably reversible.

(Slide) This slide shows a much later stage in a coronary artery. At this point you see essentially normal artery wall, the intima and media of the coronary artery and here you see a large atherosclerotic plaque which has reduced the lumen of the coronary artery to less than half its original size. In the central portion of the atheromatous plaque there is much lipid and the surface here has become fibrosed and hyalinized.

(Slide) This is another coronary artery showing a far-advanced degree of atherosclerosis. Here is the normal thickness of the vessel wall and here you can see that about 80 per cent of the lumen of this vessel is filled with a large lipid-rich, atheromatous plaque. This pale area is lipid, much of which has been removed in the staining process necessary for preparing the slide. Here is the remaining narrowed lumen of the vessel—

(Slide) And this is an even more advanced stage. In this section several of the features of an older atherosclerotic plaque may be seen,—calcification, fibrosis, and hyalinization. This is a large grumous atheroma; if this is opened up, it is found to contain soft, almost cheesy material. The lumen of this coronary is enormously reduced in size, and this narrowing of the lumen is what results in the functional disability of the organ or tissue served by this vessel.

(Slide) This is the aorta of an aging individual showing a very extensive degree of atherosclerosis. Essentially the entire aorta is composed of coalescent atherosclerotic plaques. Dr. Simms asked the question,—how does one tell early lesions from late lesions? I think perhaps the easiest way is by the color. The earlier lesions are rich in lipid and therefore yellowish in color. In the later lesions, some of the lipid has disappeared and fibrosis and hyalinization have taken place, so that the later lesions have a grayish or pearly appearance. The lesions one sees commonly in the aortas of older people are the so-called pearly plaques, the plaques that have been present for months, years, perhaps decades.

There is one aspect of atherosclerosis of the aorta that I think is related to aging.

(Slide) This is a phenomenon we are seeing with increasing frequency, an atherosclerotic aneurysm of the aorta. This is related both to age and to atherosclerosis. Atherosclerosis occurs and plaques are laid down which in the aorta do not impinge upon the lumen particularly because the aorta is such a very wide vessel, but over a period of time there is a decrease in the amount of elastic tissue in the media. Gradually, due to the intrinsic pressure within the aorta, there develops an aneurysm, usually in the lower portion of the abdominal aorta. This appears to be related to aging. It is far more common in the sixth, seventh, and eighth decades and it is becoming increasingly common. It is due, I believe, to a combination of age and the effect of atherosclerosis upon the elasticity of the aorta.

MODERATOR SIMMS: *Dr. Kellner, would you care to comment on the fatty material that is deposited in the early versus later stages, namely, the proportion of sudanophilic stainable fat as compared with the cholesterol?*

DR. KELLNER: The studies on chemical composition of atherosclerotic plaques are really not very precise. They indicate in general that the earliest plaques contain lipid which is similar in composition to the lipid that is present in the plasma. Approximately a third of the lipid is cholesterol and its ester; another third is phospholipid; and the remainder neutral fat,—essentially what one finds in the plasma. As the plaques become older and undergo various secondary changes, one finds a higher proportion of cholesterol and cholesterol ester and a smaller proportion of phospholipid and neutral fat, probably because the phospholipids and neutral fat are more soluble and tend to disappear, whereas cholesterol stays behind. I think Dr. Kendall may have something to say about that.

MODERATOR SIMMS: *Dr. Kendall, do you wish to comment further on that?*

DR. KENDALL: No, I think that I agree essentially with Dr. Kellner's statement.

MODERATOR SIMMS: *The point I had in mind in asking the question is that, particularly in the very early plaques, we certainly don't have conspicuous cholesterol deposition. There is stainable material, essentially neutral fat, whereas the cholesterol becomes more conspicuous in*

the older plaques. The old idea that atherosclerosis consists merely of cholesterol deposition is, I believe, not correct. Would you agree with that, Dr. Kellner?

DR. KELLNER: Yes. The lipid that is deposited even in the very earliest lesions is not entirely cholesterol. However, that does not imply that cholesterol may not be important in the genesis of this lesion.

MODERATOR SIMMS: *I was not referring so much to the genesis as to the actual deposition. In that connection, I might ask Dr. Kendall,—in the feeding or injection of cholesterol, what is the nature of the fat that is deposited? That is certainly not all cholesterol.*

DR. KENDALL: No. When you feed cholesterol to the animal, a rabbit or a dog, or inject a colloidal suspension of cholesterol into the plasma, although the only thing administered is cholesterol, the lipid response found in the plasma includes changes in neutral fat and phospholipids as well as cholesterol. If you inject cholesterol without any phospholipid and neutral fat into a rabbit, you will find that 24 hours later the phospholipid in the plasma of the rabbit has increased in the same relative proportions that the cholesterol has increased and with this increase in cholesterol and phospholipid there is also an increase in neutral fat. I think that workers in the field have perhaps overemphasized the part played by cholesterol and it is becoming increasingly clear, I believe, that atherosclerosis should be considered a metabolic disease involving faulty or abnormal metabolism of all the lipids rather than of cholesterol alone.

MODERATOR SIMMS: *Let us go on to the next question. Dr. Kellner has commented on the changes, the differences with the age of the lesion. Would you now make some comments on the differences in the lesions relative to age of the patient?*

DR. KELLNER: I agree with the remarks made earlier by Dr. Steele, that while atherosclerosis is more common and seen more often as age progresses, it is not inherently due to the aging process. One sees atherosclerosis early in life. One sees clinically significant and often fatal atherosclerosis in the thirties and forties, and occasionally one sees people who come to postmortem examination at age 70, 80 or even 90 with remarkably little atherosclerosis. So that atherosclerosis is not, in and of itself, the result of the aging process, but rather a disease which is superimposed upon the aging process. It is a cumulative disease. Something happens to a blood vessel which results in tissue changes

that are not removed completely. The process is additive, and what we see at the time of death is the cumulative result of all the insults that have occurred to the blood vessels over a long period of time. So that, in general, with advancing age one finds more and more atherosclerosis in the aorta, in the coronary arteries, and in the cerebral arteries. If one takes any index of atherosclerosis, one sees a fairly good relationship between age and the amount of atherosclerosis. One finds, if one takes chemical indices, that the amount of calcium or the amount of cholesterol increases with age. This, then, is a phenomenon which is associated with age, although not inherently the result of the aging process.

DR. STEELE: May I ask Dr. Kellner a question? Dr. Lansing, who is speaking here tonight, has shown a progressive change from early adult life as to the deposition of calcium and some changes in the amino acids in the vessel wall. This bears no relation to the appearance of arteriosclerosis. It seems to be a very steadily progressive affair and the calcium is in fine, microscopic particles. Do you think that has any effect on the kind of lesion, the progress of the fatty plaque that you have been talking about?

DR. KELLNER: I think that is probably a separate phenomenon from the fatty plaques, Dr. Steele. I believe that in Dr. Lansing's study he carefully chose portions of vessels that had no atherosclerotic plaques. Changes such as he described are those that appeared to be associated with the aging process rather than with the disease that we are considering today.

MODERATOR SIMMS: *I think we can go on now to a discussion of some of the clinical aspects of atherosclerosis. Dr. Steele, would you please say a few words about the recognition of the presence of atherosclerosis?*

DR. STEELE: It strikes me that the recognition of the presence of atherosclerosis is one of the main stumbling blocks to our study of the disease clinically. Many persons may have extensive atherosclerosis, with many of their arteries involved, without exhibiting clinical signs. X-rays may show a good deal of calcium here and there, particularly in the aorta, as Dr. Epstein has recently shown. But for every one picked up by clinical study there must be many more who present the disease without recognition. The autopsy rate of atherosclerosis after the age of 50 would, I think, bear this out. One misses most of the atherosclerosis that occurs unless calcium is deposited or unless it happens

to land in a strategic spot, a strategic vessel, and gives rise to symptoms. As long as it leaves the lumen of the vessel open and simply confines itself to making the wall more rigid, there is almost no way of telling of its presence. But when it encroaches upon the lumen, particularly in such spots as the coronary and cerebral vessels or the vessels of the lower extremity, then serious clinical consequences that are readily recognized supervene. One must remember, however, that it is not always atherosclerosis that blocks a coronary or cerebral artery. It is by all odds, just the most common one.

MODERATOR SIMMS: *Would you care to say a few words about the clinical treatment of atherosclerosis?*

DR. STEELE: There isn't really any specific therapy for the underlying disease. I think one must divide the treatment of atherosclerosis into the treatment of the disease itself and treatment of the consequences of vascular accidents that result from atherosclerosis. When one talks about treating atherosclerosis I am afraid that, at the moment, to outline a treatment is fraught with difficulty. Dr. Kellner has already mentioned that in the very early stages the pathologic changes in experimental disease may be reversible. Also, the late Dr. Kenneth Turner showed that deposition of the fatty plaques can be partially prevented and their resorption enhanced by the use of thyroxin. This suggests that therapy is possible. The present state of knowledge of the pathogenesis of atherosclerosis is such that I don't believe one can outline a regime which, in a real sense, is either therapy or prevention of atherosclerosis. Obviously, it seems likely that diet plays a large role. Ancel Keys' studies and those of many others suggest very strongly that there is something in the diet that may increase the rate of development of these lesions but what it is, is not clear at the moment. Certainly it is not just cholesterol that is responsible. Other kinds of fats,—animal or vegetable, may be involved. Probably the best advice is just to avoid obesity. When one comes to the treatment of the accidents that happen along the road, then that treatment is the same as it would be had the accident come from a thrombus thrown from a rheumatic heart valve or other thrombo-embolic accident. If you have a patient with hemiplegia, I don't think it makes a great deal of difference whether he is old or young. You have simply to encourage him and try and get him to move the paralyzed extremity. It is always surprising to me what simple little things can help him along the way,—a rubber ball in the

paralyzed hand, to squash it, or to encourage him to read a newspaper aloud to recover his voice. If printed words are in front of him he learns much more rapidly to speak again. Many simple procedures, often called rehabilitation, can be carried out in the home as well as elsewhere. I don't think it is desirable to go into the whole regimen of treatment of coronary thrombosis because that subject is foreign to the major purpose of this panel today.

MODERATOR SIMMS: *What would you say is the effect of atherosclerosis on the clinical picture of the elderly patient, Dr. Steele?*

DR. STEELE: I think it is much the same as the effect on younger persons. There has been considerable difference of opinion about softening of the brain which may be part of atherosclerosis and the loss of memory for recent events which is also thought to be an atherosclerotic effect on the brain. I have never been sure of this relationship because many persons who have defective memories and who are not as mentally active and vigorous as others, apparently have little atherosclerosis. Again, the difficulty in recognizing its presence is the great stumbling block to real understanding of what part it plays, for example, in what are called senile psychoses. I don't believe a similar sized thrombosis causes any greatly different result in an elderly person than in a young person. There is, of course, one very interesting observation that Dr. Blumgart made many years ago,—that if a patient has had a prior occlusion of a coronary vessel (and as we grow older we can have several), then a subsequent occlusion may not produce cardiac infarction because of the collateral circulation which has developed following the previous occlusion. In the absence of that first small occlusion, with the production of collateral circulation, the second occlusion might have caused an infarct with serious results.

MODERATOR SIMMS: *There is one more clinical question which is perhaps another way of asking the same thing. Dr. Steele, how does the presence of atherosclerosis modify the medical treatment of the patient or how is it modified because of the presence of atherosclerosis?*

DR. STEELE: I am not sure that the presence of atherosclerosis would make one modify treatment in any way. Whenever one of the accidents due to atherosclerosis occurs that really causes disability then one modifies the treatment to suit the case whether it be coronary thrombosis, cerebral thrombosis or thrombosis in the artery to the leg which might result in gangrene. In other words, it is purely the treatment for

the result of atherosclerosis that alters the way you take care of the elderly, just as you would alter the way you take care of a similar accident in a much younger person.

MODERATOR SIMMS: *Dr. Kellner, would you care to add anything to Dr. Steele's comments on the clinical side of the problem?*

DR. KELLNER: I agree with Dr. Steele. I should like to make this comment, too, that atherosclerosis, certainly clinically significant atherosclerosis, is rare in animals. Nevertheless, if they are permitted to live out their normal life spans, there is deterioration of cardiac, renal, and cerebral function which occurs in aging animals even though they do not have atherosclerosis. It may well be that many of the things we rather glibly call atherosclerotic changes in the brain are not due to atherosclerosis but rather to some other phenomenon of aging.

MODERATOR SIMMS: We have been snowed under with questions so I think we had better start dealing with them. There are additional subjects on our agenda regarding the etiology of atherosclerosis and following that we will take up as many as possible of these questions from the audience. I don't think we will be able to cover them all by any means.

As to the etiology of this condition I am going to ask Dr. Kendall to comment on the current theories.

DR. KENDALL: There is almost universal acceptance today of the idea that some metabolic defect is the cause of atherosclerosis. There is no agreement whatsoever on what this metabolic defect is. It is believed by some that this metabolic defect is manifested by an increase in the beta-lipoprotein levels in human beings. I specify beta-lipoproteins rather than cholesterol levels because the bulk of the evidence today seems to indicate that the beta-lipoproteins play a special role in the development of the arterial lesions, a more specific role than does the serum cholesterol taken as an entity. For instance, the normal human being will have an average serum cholesterol level of about 200 mg. per cent. The average dog also has a serum cholesterol level of 200 mg. per cent but in the human being about 150 mg. of this 200 is present in the beta-lipoprotein fraction. In the dog only 40 to 50 mg. is present in the beta fraction. If by feeding cholesterol we increase the serum cholesterol level in a dog to 400 mg. per cent, a level which we might expect to produce atherosclerosis, we find there is no change in the ratio between the alpha and beta-lipoproteins. The cholesterol in the

beta fraction only reaches 80 to 100 mg. per cent, a value still much lower than the level of beta-lipoprotein normally found in most normal human beings. Under these conditions the dog does not develop atheromatous plaques. However, if we can maintain the serum cholesterol level of the dog at 400 to 500 mg. per cent and at the same time produce a change in the distribution of the cholesterol between the alpha and beta fractions, so that the dog now has 150 to 200 mg. per cent of cholesterol in the beta fraction, the dog will develop atheromata. Similar concentrations of beta-lipoprotein will produce arterial lesions in other experimental animals. For instance, normally a rabbit transports practically all of his cholesterol in the alpha fraction. But, if by feeding cholesterol or by any other means the level of the beta-lipoprotein is increased up to the level commonly seen in human beings, atheromata result. So it appears to be as though human beings have a metabolic pattern different from that found in animals, which results in the maintenance of the beta-lipoprotein at a point where atherosclerosis tends to develop. It may be that in clinical disease this abnormally high beta-lipoprotein level is superimposed upon other factors, which cause local damage to certain portions of the arterial tree. I am thinking particularly of the significant work done by Waters and de Suto-Nagy at Yale. They have shown that young dogs injected with certain pressor amines, including epinephrine, develop lesions in their coronary arteries and nowhere else. These lesions are not atheromatous. They would be described as arteritis. But if the concentration of beta-lipoprotein is increased, the lesions assume the appearance of typical atheroma.

I would say that to the best of my belief at present, atherosclerosis is a result of an elevated beta-lipoprotein level, perhaps combined with other localizing factors. I must point out that this elevated beta-lipoprotein level is not in itself a metabolic defect. It is the result of a process that is taking place elsewhere. The level of any component in the blood is determined not only by what is taking place in the blood but also by what is happening in the cells of the body. I believe the basic metabolic defect that leads to the elevated beta globulin is a defect that lies in the inability of the peripheral cells to take up and utilize the lipids which are supplied to them in the plasma. Whether we can pinpoint this defect in the future remains to be seen. There are certain bits of evidence at hand now that show that progress is being made.

One, it has been shown that the injection of heparin will increase

the individual's ability to clear lipid from the plasma. It has been shown that the clearing factor that appears in the blood is a lipase that splits the neutral fats of beta-lipoproteins. But this lipase originates in the cells. The effect of heparin is to liberate the lipase from the cells and permit it to get into the plasma and I am not at all certain that the appearance of this lipase in the plasma is an important factor. I think perhaps the presence of the lipase in the peripheral cells is the important thing. A cell has to have this lipase in order to begin the metabolic oxidation of fatty acids. Perhaps the heparin exerts its influence by modifying the surface of the cells. Heparin binds calcium. Calcium ions are necessary for the maintenance of the integrity of a cell wall. The appearance of the clearing factor in the circulating plasma may be the result of changes in the cell walls caused by the presence of an excessive amount of heparin that permits the lipase normally present within the cells to spill out into the blood. *This is speculation, which at present does not deserve to be given the name of a hypothesis, to say nothing of a theory.*

A far more significant observation, so far as the atherosclerosis is concerned, is the observation that patients with severe atherosclerosis produce less clearing factor in their blood following the injection of heparin. This may indicate that these individuals lack lipase in their peripheral cells.

All of us in this field are groping for some clue to the basic mechanism that results in the elevation of the beta-lipoprotein. Other clues are perhaps being furnished by the hormones. We know that various hormonal imbalances will produce marked changes in the serum lipid pattern, and I speak of hormonal imbalance rather than the effect of any specific hormone because I believe that it is impossible to introduce excessive amounts of any one hormone into a living animal without producing compensatory effects upon the complete hormonal balance that regulates metabolic processes in the body. One cannot affect the pituitary without affecting the thyroid and the adrenals and the pancreas. One cannot affect the gonads without producing compensatory effects in these other glands. So I think it is probably too early to discuss hormonal therapy in atherosclerosis intelligently. We do know that the only hormones which have been demonstrated to produce shifts in the alpha and beta-lipoprotein fractions are estrogens and androgens. Estrogens will tend to shift the cholesterol from the beta

to the alpha fraction. Androgens tend to produce shifts in the opposite direction. I think these shifts from one fraction to another are far more important than shifts in the level of serum cholesterol.

Much has been said in recent years about the effect of diet, about the possibility that high fat diets may play a part in the etiology of this condition. Much of the evidence that has been presented is rather convincing and I would be tempted to say that probably the high beta-lipoprotein concentration in human beings may be the result of an imbalanced hormonal picture upon an individual who is eating an unbalanced diet.

MODERATOR SIMMS: Dr. Kendall has given us a picture of the etiology of atherosclerosis but, as he pointed out in the beginning, there are many theories and many individuals working in the field who do not altogether agree with each other. So I think Dr. Kendall will agree that there are others who may not entirely subscribe to the picture which he has presented. My own work, for example, indicates that the important materials are not necessarily lipoproteins but other lipoid materials.

There are so many questions from the audience that I think we should turn our meeting over to an attempt to answer some of them. I regret that we will not be able to cover all of them.

First, there is a short but important question as to the relation of atherosclerosis to hypertension. Dr. Kellner, would you try to answer that?

DR. KELLNER: I think that atherosclerosis does not cause hypertension, and conversely, I do not believe that hypertension causes atherosclerosis. However, I do believe that hypertension has an important effect upon the development of atherosclerosis, an important accelerating effect. Lipids permeate the walls of blood vessels and the higher the inherent pressure in the vessel, the more lipid will permeate. In general, the higher the pressure, the more rapid and the more severe the development of atherosclerosis. I think this is true both in experimental animals and in human beings. Individuals with hypertension are more prone to develop clinically significant atherosclerosis of the coronary arteries and cerebral arteries. For example, females under the age of 40, who rarely get coronary artery sclerosis, do so if they have high blood pressure. If atherosclerosis results from the metabolic defect which Dr. Kendall has beautifully described, you might ask why does it not occur in veins?

Why does it not occur more commonly in the pulmonary artery? The same lipids are present in the veins as are present in the arteries. I think the answer, in part at least, is the pressure in these vessels. The vessels with the higher pressure are more apt to develop atherosclerosis. Veins which have little pressure have little or no atherosclerosis. The pulmonary artery which normally has a low pressure usually has little atherosclerosis. However, in those instances where the pulmonary artery pressure is elevated, as in mitral stenosis, one sees much more atherosclerosis. In the experimental animal, too, Dr. Kendall and his associates have shown that if one feeds dogs cholesterol and alters thyroid function by thiouracil, the animals develop hyperlipemia or hyperbetaglobulinemia and eventually develop atherosclerosis. If, in addition, the animals are made hypertensive and the lipid metabolic defect which has been shown to produce atherosclerosis in the dog is added, then the animals develop atherosclerosis very much more rapidly. To sum up, hypertension increases the speed with which atherosclerosis develops in experimental animals and probably also in human beings.

MODERATOR SIMMS: *Here is a question in regard to heparin administration: Is heparin administration of any value in the prevention of atherosclerosis? Dr. Steele, would you care to comment?*

DR. STEELE: I have just learned from Dr. Kendall a good deal about the action of heparin. Its administration might be useful if the beta-lipoproteins are so important in the development of the disease. Its administration is rather impracticable in long drawn out illnesses and one actually does not know that reducing the level of these lipoproteins would ameliorate arteriosclerosis.

MODERATE SIMMS: *Here is a question in regard to salt. What is the role of salt in the diet in atherosclerosis?*

DR. STEELE: I should not think it had any role. I don't know whether any definitive experiments have been done on the subject but certainly one must control the intake of salt when there is a disturbed electrolyte balance and the patient is accumulating fluid. Therefore in arteriosclerotic heart disease, with congestive failure one would probably want to restrict salt. Also some persons with arterial hypertension are able to stay on a very limited salt diet for long periods and can reduce the level of blood pressure somewhat. Insofar as hypertension is a factor in the development of atherosclerosis, reduction of pressure levels might aid in prevention.

MODERATOR SIMMS: *What would you say is the role of iodides in the treatment of atherosclerosis?*

DR. STEELE: I like that question! In the latter part of the last century when I was a youngster and my father was a practitioner of medicine, iodide was one of the really fine cure-alls for everything from 40 years of age on up. Iodides were given in courses of gradually increasing doses so as not to upset the stomach, in order to get the dose up as high as possible and then drop off again. It was supposedly useful in dissolution of the deposition of the fatty gumma of syphilis. It was similarly thought that one might dissolve the fat of the fatty plaques of arterial disease. A good many of my colleagues look askance at me when I give large doses of iodides today. It seems to be a favorite medicine of too long ago for anyone to use now. This is a purely clinical impression, but I do believe that some patients suffering from tinnitus and dizziness have been benefited by the use of iodides. Clinical improvement could be explained on the basis that a small early plaque in the pterygoid artery to the ear, which is enclosed in bone with no room to expand, had been dissolved or reduced in size. The evidence is very thin.

MODERATOR SIMMS: *Here is a question about an apparent disagreement between Dr. Kellner and myself in regard to the amount of cholesterol in the early plaques as compared with the later plaques. What I attempted to say was that in the early pin-point plaques, as far as the microscopic examination is concerned, the neutral, stainable fats predominate. One does not see cholesterol crystals, whereas the cholesterol crystals appear in the later plaques. Also, I might refer to Jobling and Meeker's work in which they found on chemical analysis that the proportion of cholesterol in the plaques is less in the early plaques than in the later ones.*

Now, Dr. Kellner, is there any disagreement between those comments and your opinion?

DR. KELLNER: I don't believe there is any disagreement at all, Dr. Simms.

MODERATOR SIMMS: *Does the ingestion of eggs influence atherosclerosis? Who wants to take that?*

DR. STEELE: For a great many years Dr. Steiner, who has worked with Dr. Kendall, has tried to alter the cholesterol level by feeding yolk of eggs to patients and Dr. Messenger and myself worked pretty hard at it too. It is surprising how many eggs you must eat before you

even begin to get an effect on the blood cholesterol level, after weeks and months of such feeding. Even then, although the levels ran a little bit higher, they were not much to brag about. However, Dr. Steiner did show that the cholesterol level often does run higher and is much more variable in persons with coronary disease. Although you probably would not want to try the experiment, if you feed very large amounts of eggs to people who have coronary atherosclerosis it might be that they will be less able to control their cholesterol level than normal people. Eggs also have a large amount of lecithin and where the cholesterol-lecithin ratio appears to be lower, as Pomerantz and Kunkel showed in a large group of diabetics, atherosclerosis is less likely to occur. If you manage to build up the blood phospholipids to a greater extent than the cholesterol, the eggs may well be good for you.

MODERATOR SIMMS: *Dr. Kendall, here is one for you. What significance do you attach to the lipoprotein spectrum of the blood in regard to the presence of atherosclerosis? I assume the person who asked this question is referring to Gofman's Sf* fractions, which Gofman claims do increase in atherosclerosis, particularly in the Sf20-400 fraction.*

DR. KENDALL: I find that a hard question to answer. I believe that the difficulty in answering questions of this kind arises from the fact that most adult Americans have some degree of arteriosclerosis. Just because an individual has not yet had a coronary attack, or has not developed a plaque in a position where it gives him symptoms is no reason for believing that the process leading to atheromata is lacking in this individual. I think that the Sf spectrum, lipoprotein spectrum of Gofman, will give you information about the relative height of the beta-lipoprotein level but I don't think it gives information that is much more valuable to the clinician than knowledge of the total serum cholesterol level. If the beta-lipoprotein is elevated to such an extent that it produces a significant change in the Gofman fraction it is going to be elevated enough to be significant in the serum cholesterol levels. Just as we do not know how to interpret the significance of a serum cholesterol value of 300 mg. per cent as compared with 250 mg. per cent, I am at a loss to compare an Sf12-20 level of 48 with one of 34. I think that the work on the ultracentrifuge is stimulating to this field but this work is still in the experimental investigational phase and we cannot apply it to individuals.

* Svedberg unit = 10^{-13} cm./sec./dyne/gm.

MODERATOR SIMMS: Dr. Kendall has touched on a serious research difficulty in atherosclerosis, namely finding or selecting individuals who have a propensity for atherosclerosis. If a patient has had a coronary attack we are dealing with someone who did have a propensity but we don't know that he has it at that time until after the attack particularly if he is under proper treatment. So we are handicapped in attempting to find a correlation between any measure of propensity toward atherosclerosis and the clinical picture in the patient.

I have a question which I will ask Dr. Kellner to discuss,—the difference between atherosclerosis and arteriosclerosis.

DR. KELLNER: Arteriosclerosis or hardening of the arteries is a general term, a term which includes within it more than one disease of blood vessels. Under the heading of arteriosclerosis we include, for example, atherosclerosis, the disease we have been discussing this morning. Another type of arteriosclerosis is the Mönckeberg type of sclerosis, which occurs usually in the lower extremities, which affects the media of blood vessels rather than the intima resulting in calcification, and which by itself does not narrow the lumen of the vessel. Another type of arteriosclerosis might be the change that occurs in very small blood vessels associated with hypertension. Yet a fourth type of arteriosclerosis might be the type of change that occurs with aging, where the elastic tissue of blood vessels undergoes degenerative changes and the vessels dilate and become tortuous, as can readily be seen in the corkscrew temporal arteries of most people in middle life. This is neither Mönckeberg sclerosis nor atherosclerosis, but a weakening of the elastic fibers of the vessel wall so that the wall undergoes dilatation, in both the longitudinal and transvere directions. Arteriosclerosis, then, is the generic term which refers to many different diseases of blood vessels. Atherosclerosis, we hope refers to a single specific entity among vascular diseases.

MODERATOR SIMMS: It is now time to close the meeting. If any of you wish to stay longer and discuss other questions personally, the panel members will be glad to do so. The meeting is adjourned.

MEETING COMMEMORATING THE CENTENARY OF FLORENCE NIGHTINGALE IN THE CRIMEA

HELD ON MAY 11, 1955 BY THE

SECTION ON HISTORICAL AND CULTURAL MEDICINE OF THE NEW YORK ACADEMY OF MEDICINE

FLORENCE NIGHTINGALE'S PLACE IN BRITISH HISTORY

F. B. A. RUNDALL, C.M.G., O.B.E.*

British Consul General in New York

FLORENCE NIGHTINGALE'S INFLUENCE ON NURSING

MARGARET G. ARNSTEIN, R.N., M.P.H., D.SC.

Chief of the Division of Nursing Resources, United States Public Health Service

FLORENCE NIGHTINGALE'S INFLUENCE ON MILITARY MEDICINE

FRANK B. BERRY, M.D.

Assistant Secretary of Defense, Health and Medical Affairs, Washington, D. C.

Portrait from the *Florence Nightingale Collection*, School of Nursing, Presbyterian Hospital, N. Y.

* Now Sir Francis Rundall, K.C.M.G., O.B.E.

FLORENCE NIGHTINGALE'S PLACE IN BRITISH HISTORY

F. B. A. Rundall, C.M.G., O.B.E.

British Consul General in New York

I am honoured at being asked to read the first paper this evening at your meeting to celebrate Florence Nightingale's inspection of the British army hospitals in the Crimea a hundred years ago. My subject is Florence Nightingale's place in British history, and I propose to try to put her life and work in its historical perspective. I shall leave it to the distinguished speakers who follow me to fill in the details of her career and to describe to you the great events in which she played so prominent a part. I hope you will forgive me if much of what I say sounds superficial; it is difficult to cover so broad a field in the time at our disposal.

People tend to think of Florence Nightingale as an historical figure who lived a hundred years ago. This is of course true, but it is also true—and not many people realize it—that she was still alive during the lifetime of many of us. Florence Nightingale was born in 1820 and died in 1910. Her 90 years covered perhaps the most momentous period of change and development in British history. It is interesting to contrast the two dates. In the year of her birth King George the Third was still on the throne. The overthrow of Napoleon at Waterloo, which marked the end of the series of wars which raged through Europe—and in this country also—had happened only five years before. Britain was still, in all but chronology, in the 18th Century. Transportation moved at the pace of coach and canal; the industrial revolution had hardly begun and had not yet shown either its good or ill effects. England was still ruled by the aristocracy, the peers and great country squires who had no doubt whatever of their divine right to do so. The great Reform Bill of 1832, which abolished what were called the rotten boroughs and restored to the ordinary citizen his power to elect the representatives of his choice to Parliament, was yet to come, although the social unrest which led to its passage was already apparent. In foreign affairs

we had peace, an uneasy peace to be sure, but one which was to last for nearly a 100 years and would be broken for us only by the Crimean War and the Indian Mutiny.

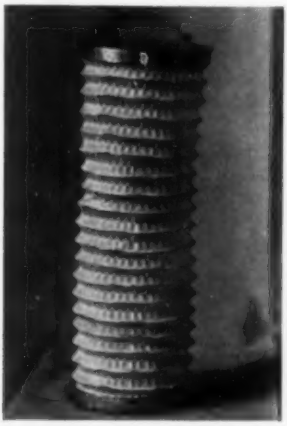
Florence Nightingale died in 1910, and four years later the century of the *pax Britannica* came to an end. With all its faults—and they were many—this was one of the great periods of British history. The British Empire expanded and prospered under cover of the peace imposed upon the world by the power of its navy. British trade followed the flag all over the world. Profiting from her great industries of coal, steel and textiles, England became the industrial and trading centre of the world. Her surplus wealth, invested overseas for no better reason than the greater profit of the investors, helped to open up the United States, Canada, South America and many other areas besides.

When Florence Nightingale died—and she had been out of touch with the outside world for some time before her death—Queen Victoria was already a memory. The brief and brilliant flowering of the Edwardian era was passing under the ominous clouds of the First World War.

What part did this extraordinary, selfless, obstinate, exhausted and dedicated figure play in her rich period of history? I think that one can fairly claim that she was responsible to a great extent for two very significant developments. It would be an exaggeration to say that she was the catalyst of the 19th Century. In one field directly, and in another indirectly, the long-term consequences of her comparatively short life, when she was in the public eye were profound indeed.

I am sure that Dr. Berry will deal with Florence Nightingale's influence on the British army, and specifically upon its medical and supply services. The changes she brought about were indeed great, although the most essential army reform, the abolition of entry and promotion among its officers by the purchase of their commissions, did not come until 1870, when the Franco-Prussian War and the rise of Germany made the British people look once more to their defences. But in this long-term view that we are taking it is not so much what Florence Nightingale did, which was great, but the publicity she caused, which had the greatest effect.

Dr. Berry will tell us about the Crimean War and how the British army, the invincibles of Waterloo, were on the edge of defeat by starvation, pestilence and the stupidity of their leaders. In that war Florence Nightingale became the symbol of humanity and reform. She could



The famous lamp which Florence Nightingale carried around the wards at Scutari. The lamp is now in the United Service Museum in London.

not have done her work, however, without the bright light of publicity thrown upon her by the first foreign war correspondent, William Howard Russell of the "London Times," and it was the public indignation aroused by Russell's despatches that enabled her to succeed.

Florence Nightingale was indeed both a product and a pioneer of the new sentiment of humanity and consideration for their fellow-men which marked the early Victorians. This sentiment is reflected in the novels of Dickens, in Charles Kingsley's "Water Babies" and the acts of Parliament that were eventually passed to limit hours of work and to control the labour in mines and factories of women and children.

There is no doubt that this newly aroused public opinion was, in the main, an expression of the feelings of the middle and lower classes of society. But 18th Century England was ruled by its aristocracy, who had returned to power with the restoration of King Charles the Second towards the end of the 17th Century determined to stand no nonsense from the doctrinaire followers of Cromwell. By the time of the Crimea the aristocracy were already on the defensive. The Reform Bill of 1832 had removed a great deal of their political power. The Irish potato famine of 1848 and the ghastly suffering it caused had showed many of them up as inefficient and heartless landlords. It took the Crimean War to reveal them as superlatively courageous but impossibly stupid military leaders. If one may paraphrase Oscar Wilde, the British army at that time were the indomitable led (if one could so call it) by the impossible. You need only read Cecil Woodham Smith's great

book "The Reason Why," about the events leading up to the Charge of the Light Brigade, to appreciate the unbelievable conditions which prevailed.

It can be claimed then that Florence Nightingale's work and the publicity given to it was one more important nail in the coffin of aristocratic rule. One of her chief objectives both during and after the war was to ameliorate the lot of the common soldier. Russell's despatches showed him to be a hero; Florence Nightingale saw to it that he reaped some of the fruits of his heroism.

Florence Nightingale's second great long-term accomplishment was the emancipation of women. I must be careful here not to trespass on Dr. Arnstein's territory, but Florence Nightingale first came into the public eye in 1853, when she was appointed lady superintendent of the newly-opened establishment for Gentlewomen During Illness. This was one of the first free hospitals recognizing the need of poor members of the upper and middle classes. It is still going and now bears Miss Nightingale's name, although its inmates can no longer expect to pay only $3.00 a week for a single room and $1.50 a week for a double.

Perhaps as a result of her experiences in this hospital Miss Nightingale's motivation during the Crimean War was almost as much to prove that nursing was a profession for gentlewomen, instead of gin-swilling Sairy Gamps, as to relieve the distress of the troops. With immense difficulty she proved her point, and what is more, proved it to a world audience. It is hard for us to realize that before Florence Nightingale the idea that a woman could be self-supporting except in domestic service—or worse—was not seriously contemplated.

And so what happened? Well, John Stuart Mills' great book "The Subjection of Women," written in support of women's suffrage, appeared in 1869. In the latter half of the 19th Century women's colleges, an unheard of thing, were founded at both Oxford and Cambridge. Women's suffrage had to wait until the 20th Century, but the right of women to have brains, to cultivate them and to support themselves in the learned and other professions was slowly established. When we in England think of the part that women played to defend our country during the last war, and marvel at it, we should also think back to the pioneer of them all, Florence Nightingale. Living quietly in her little house in Westminster during her old age, Miss Nightingale neither knew nor greatly cared, but she had sparked a social revolution.

FLORENCE NIGHTINGALE'S INFLUENCE ON NURSING

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MODERN nursing derives so completely from the example and teaching of Florence Nightingale that it is hard to pick out the particular practices that owe their existence to her influence. All nursing has been influenced by her. One might say modern nursing is Miss Nightingale—that her name is a synonym for nursing. She demonstrated in a dramatic fashion in the Crimea that nursing—and sanitation—could reduce mortality, as Dr. Berry will describe to you.

The medical profession has long recognized the essential role of nursing in care of patients and prevention of illness. The Academy of Medicine tonight in paying tribute to Florence Nightingale is paying tribute to the contribution nursing makes to medical practice.

Nursing literally did not exist except in a few religious orders until Miss Nightingale showed what it could do, and more important, established a school to produce people who could do it. This first school at St. Thomas' Hospital in London was a model for all the later schools in England and the United States. Nightingale nurses became heads of all the early schools established in England. Bellevue Hospital School of Nursing in New York, the first in this country to introduce the Nightingale principles, was actually guided by letters from Miss Nightingale.

This evening I would like to detach Miss Nightingale's principles from their historical setting and discuss them in relation to modern nursing. To do this one must recognize that Miss Nightingale had both a good influence and a bad influence on nursing. Most of the "bad" is due to the fact that we have slavishly followed some practices she strongly advocated, long after the need for them has disappeared. Some of it is due to the fact that we have continued with practices she initiated, but have ignored the underlying principles which she set forth.

The influences that have outlived their social usefulness are almost entirely related to the conduct of student nurses. When Miss Nightingale started the first training school, the Nightingale nurse, in the words of one of her biographers, "had to establish her character in a profession proverbial for its immorality."

The probationers, as students were called, had their entire lives controlled by the school as though in a convent. They had two hours of outdoor exercise each day, outside the dormitory. They were never allowed to leave the dormitory alone. All other time was scheduled: meals, work and learning, and sleep. All this control probably was necessary at *that* time. The fact that some of it has lingered on into the middle of the Twentieth Century shows we did not consider the reason for the practice but clung to the practice itself long after the respectability of nursing was established, and young women in our society had attained much more independence.

Miss Nightingale herself recognized the danger of stereotyping. In the paper, "Sick Nursing and Health Nursing," that she read in Chicago in 1893, when she was in her seventies, she said, "No system can endure that does not march."

On the other hand, if we had followed as closely all else that she taught, we would not now be trying to undo so much of what we recently have been teaching and doing in this country in nursing.

This astonishing person had such vision and understanding that I sometimes think a school of nursing today could not do better than read from her writings each morning a "lesson for the day." The lesson could then be expanded with the knowledge of human relations and with the scientific facts we have acquired since her day, but I doubt that many "new lessons" would be needed.

It is a little disconcerting to realize some of our most modern ideas—that are still opposed by some nurses and some of the related professions—that these dangerous "new" ideas were taught by Miss Nightingale 95 years ago. True, our leaders in nursing today have come to these ideas quite independently and have contributed greatly to getting us "back on the track" and several miles ahead of where we went off.

We might consider these ideas under three main headings: care of the patient in the hospital; administration of nursing services; and education of nurses.

About care of the patient, the most advanced members of the nurs-

ing profession are stressing the idea that we must nurse the whole patient and not just his disease. Miss Nightingale expressed the same principle. She said, "the art is that of nursing the sick." And she added, "Please mark—nursing *the sick;* not nursing sickness."

Her definition of nursing the sick included giving the medicines and stimulants prescribed, and the surgical appliances, proper use of fresh air, warmth and cleanliness, proper choosing and giving the diet, and quiet. All this was to be done, she wrote, "at least expense of vital power of the sick."

Psychosomatic medicine is a relatively new concept, yet the great physicians and nurses of the past recognized the influence of emotions on disease conditions. In writing about the care of patients, Miss Nightingale gave attention to the ways a patient's illness is affected by his state of mind, his worries and fears, the attractiveness or drabness of his surroundings, and the noise around him. She summed it up as follows:

"The symptoms or the sufferings generally considered to be inevitable and incident to the disease are very often not symptoms of the disease at all, but of something quite different—of the want of fresh air . . . or of quiet or of cleanliness or punctuality. . . . The reparative process . . . has been hindered . . . by some want in one or in all of these things. . . ."

We can almost hear the modern cardiologist saying as she did, "Apprehension, uncertainty, waiting, expectation, fear of surprise, do a patient more harm than any exertion."

Psychiatrists today teach us that the type of reassurance which tells a patient not to worry, the operation will not amount to anything, he will be all right, and other such encouragements, often defeats its own end. Today we believe that listening to the patient, the process we call non-directive interviewing, is actually more helpful. Although we did not learn this principle from Florence Nightingale, we might have done so. Although no one had ever heard of "non-directive interviewing", Miss Nightingale wrote: "Do not cheer the sick by making light of their danger", and continued at length to discuss the understanding of patients' fears and their individual differences.

In recent years we have recognized that color affects the productivity of industrial workers, and are now experimenting with the effect of color on patients. Although Miss Nightingale made no controlled experiments, she observed keenly, and in this instance her observations have been proved correct by later scientific experiments. "Little as we

know about the way in which we are affected by form, by colour, and light", she wrote, "we do know this, they have actual physical effect." Another observation from her writing of the same period was: "No one who has watched the sick can doubt the fact, that some feel stimulus from looking at scarlet flowers, exhaustion from looking at deep blue, etc."

On the other hand, Miss Nightingale recognized that the connection between mind and body is not a one-way street. She said she wished "a little more was thought of the effect of the body on the mind." Today we recognize this fact and no longer say as we did some years ago that a patient "is or is not cooperative." We try to understand *why* he complains, or why he is angry, or why he resists treatment. Miss Nightingale summed up the patient's difficulties so succinctly that we might repeat her words every day: "Almost *any* sick person, who behaves decently well, exercises more self-control every moment of his day than you will ever know till you are sick yourself."

Until recently, the recognition given Florence Nightingale by the nursing profession has been primarily for her work in establishing decent standards of nursing care for patients and starting a system of training for those who give the care. Yet perhaps her real genius was in the management field. She may have been so far ahead of her time that we did not appreciate the principles she bequeathed us; we have had to "discover" them for ourselves. Nurses, like hospital administrators, have gone to industry and business for ideas on better management. So we cannot say that Miss Nightingale has influenced our profession greatly in this aspect of nursing service. Yet she knew the basic principles of good management and expressed them so well I would like to quote part of her statement on the first requirement of an administrator:

"To be 'in charge' is certainly not only to carry out the proper measures yourself but to see that everyone else does so too. . . . It is neither to do everything yourself nor to appoint a number of people to the [same] duty. . . ."

Miss Nightingale had much to say about applying this principle and other principles of good management. In 1858 she advocated conserving the nurse's time and energy in almost the exact same words that were used in 1955 in a recommendation growing out of a recent time study of nursing activities. I quote one sentence from the report by a Michigan hospital:

"Following the study made in our hospital we now have a messenger service that brings supplies and drugs to the patient units and transports patients from place to place in the hospital."

Miss Nightingale said that "nothing should be fetched by the nurses", and that "the nurse should never be obliged to quit her floor except for her own dinner and supper." She had schemes to make this possible—more revolutionary in her day than pneumatic tubes in ours—a "windlass installation" or lift to bring up the patients' food. She also had a scheme for saving work by having hot water "piped up to every floor." Without systems of this kind, she said, the nurse is converted into a pair of legs.

She was not only an administrator but an inventor of labor-saving devices. The intercommunicating system at which we marvel today seems only a natural descendant of her suggestion that "the bells of patients should all ring in the passage outside the nurse's door *on that story* and should have a valve which flies open when its bell rings and *remains* open in order that the nurse may see who has rung."

Perhaps some of Miss Nightingale's ideas have remained dormant in nurses' consciousness like recessive characteristics. Even today only the more progressive hospitals have written job descriptions for all nursing personnel. We rather timidly suggest that the good staff nurse should receive recognition through higher pay and more responsibility without necessarily having to become an administrator.

Miss Nightingale said in 1858 in her "Subsidiary Notes as to the Introduction of Female Nursing Into Military Hospitals" that the duties of each nurse, senior and junior, and of the orderly should be clearly outlined. She also wrote: "Many women are valuable as nurses who are yet unfit for promotion to head nurses. It appears to me that it would be desirable to have intermediate recompense. . . ."

The recent studies to which I referred have analyzed the pattern of interruptions in the head nurse's activities and have shown that the head nurse seldom spends as much as five minutes on an activity without being interrupted. Miss Nightingale had a gloomy outlook for people who spent their days in this fashion. She said: "I have never known persons who exposed themselves for years to constant interruption who did not muddle away their intellects by it at last."

Examples of her astute understanding of administration and her inventiveness could be multiplied for the rest of the evening. I shall

quote only one more, under the heading of administration: "Unless the Matron's authority is supported by the Principal Medical Officer, the patients always suffer." When we read Miss Nightingale's writings we are never in danger of forgetting the patient.

In the field of nursing education, we have been grappling with numerous perplexing problems and divergent ideas in recent years. Here again Miss Nightingale offered many ideas that today would be considered progressive, perhaps even radical. Many students of our system of nursing education maintain that the school of nursing should be independent of the hospital. It is interesting to note that the first school of nursing, the Nightingale School at St. Thomas' Hospital in London, had its own endowment fund and its own board of managers.

The first schools in this country, at Bellevue and Massachusetts General Hospitals, were not established primarily to ensure better care of the sick in those hospitals but to educate nurses. The impetus for these schools came not from the physicians or the hospitals but from the New York State Charities Aid Association and from the Women's Education Association in Boston. Each was directed initially by its own board of managers.

Recent studies of costs of schools of nursing have wrestled with the question, "Should nursing service personnel, head nurses and others be paid by the school for their contribution to the education of the students?" In the Nightingale school, the head nurses and the director of nurses were paid part of their salaries from the Nightingale fund for the training of students. So we at least have a precedent for this practice.

Some of us have believed that having the students work full time on the wards was "the good old way." Yet even in Miss Nightingale's era, when theory was a much smaller part of the preparation for any profession than it is now, Miss Nightingale stated as one of the essentials of a training school that "there shall be an organization which by giving proper help in the wards gives probationers time to do their work as pupils" as well as give service to patients. Seventy years later we are proposing not a completely different philosophy of training, but rather more of the pupil and less of the service.

Nursing educators are convinced of the necessity of students understanding the reasons behind the things they do—of the value of the case-study method of teaching. Miss Nightingale must be nodding her

head in approval. Her spirit perhaps is murmuring "at last." For in 1882 she wrote, "We require a special organization for the purpose of training." Then she explained:

"Training is to teach not only what is to be done . . . not only how to do it, but *why* such and such a thing is done, and not such and such another; as also to teach symptoms, and what symptoms indicate what of a disease or change, and the 'reason why' of such symptoms.

"Without time for these things, average nurse-probationers degenerate into conceited ward-drudges. Without a system for these things, they potter and cobble out their year about the patients, and make not much progress in real nursing—that is, in obeying the physicians' and surgeons' orders intelligently and perfectly."

In her forthright way, Miss Nightingale predicted dire things for the students if we do not give them proper training.

She summarized so well all the attributes we want in a nurse today that I am going to end with this quotation:

"Training is to teach a nurse to know her business, that is, to observe exactly, to understand, to know exactly, to do, to tell exactly, in such stupendous issues as life and death, health and disease.

"Training is to enable the nurse to act for the best in carrying out her orders, not as a machine but as a nurse; as an intelligent and responsible being. Training has to make her, not servile, but loyal to medical orders and authorities. True loyalty to orders cannot be without the independent sense of responsibility, which alone secures real trustworthiness."

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FLORENCE NIGHTINGALE'S INFLUENCE ON MILITARY MEDICINE

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THE Duke of Wellington described his army which won the victories in the Peninsula and at Waterloo as "the scum of the earth enlisted for drink." After the Battle of Waterloo, the old order continued to prevail. There was a complete separation between officers and men and little, if any, interest by the former in the well-being of the latter. The same neglect on the part of the officers had existed in the Peninsula campaign and before but the public had been kept in complete ignorance. It was only due to the fact that an Irish correspondent of the London Times, William H. Russell, reported the conditions as they were in the Crimea and in Scutari in the fall of 1854, that the British War Office requested Miss Florence Nightingale to go there with nurses. Simultaneously with this request, she had already made her own plans to take a small group there. The War Office and everything emanating from it was bound by restricted and outmoded forms and paralyzing red tape. For example, there were a Secretary for War and a Secretary at War, and in the supply of the Army and hospitals there were a Commissary General and a Purveyor General. The duties of the former consisted of actual purchase of supplies and then it was the function of the latter to deliver them to the hospitals. Men were supposed to arrive at the hospitals fully clothed and with extra sets of clothing. When this was not the case, as is usual after battle, there was nothing that could be done about it, for the purveyor could not issue new clothing. The medical officers in general thought of little except their own protection so that as long as they made out the necessary forms and requisitions, they were free from blame and the fact that they had nothing to work with concerned them little if at all. Again, for the most part, their own knowledge was limited and their interest in the welfare of the men somewhat less. Commissions were still sold in the British army whether or not those who purchased them had any train-

ing or knew anything about the military. This wretched system had been defended by the Old Duke and his influence was paramount up to the time of his death only two years before the onset of the Crimean War. In the years following Waterloo, a narrow, parsimonious policy had been followed, with no attempt to keep the army in modern condition or with modern organization as is so common with other nations following wars and great periods of letdown and lethargy; even some of the muskets with which the troops were supplied were those that had been used at Waterloo and in the Peninsula. In 1854 the Commissary General was a veteran of the Peninsula and the Purveyor General who accompanied the troops to the Crimea was a veteran 70 years of age. Dr. Hall, the Chief Surgeon who had been ordered up from India, was disappointed that he had not been permitted to proceed to England first and throughout the Crimean Campaign never hesitated to show his displeasure and ruthlessness towards the troops, his own medical staff and the Nightingale group of nurses.

In order to avoid favoritism, Miss Nightingale recruited her nurses without regard to religious beliefs, so that two groups of Roman Catholics, another from the High Anglican Church, and others from the Low Church made up the group. Later Miss Mary Stanley brought an intensely Catholic group with her from England without authority of Miss Nightingale, to serve in a dual role, nursing and ecclesiastical. The result was trouble from the very first and Miss Nightingale was assailed from both sides. Fortunately, however, she had taken with her a large sum of money provided by personal friends and from the fund raised by the London Times and this plus her orders gave her complete freedom of action so that whatever was lacking could be swiftly provided by her in the open market. But a flaw in her orders caused untold trouble. The original letter from the War Office stated that she was to be in charge of the nurses services in Turkey, and it was not until over a year later that this was corrected and her authority extended over the whole nursing services and nurses in the Crimea. Owing to the hostility between her and Dr. Hall and his refusal to accept her or any of her ideas, although she could go to the Crimea, little attention was paid to her there and she was treated with every discourtesy. This was carried to such limits that in the spring of 1855, when she was taken extremely ill while there, secret arrangements were made by Dr. Hall to put her on a ship which went directly to England without

stopping at Scutari to put her off at her own hospital. Fortunately, however, this plot was discovered in time and she was transferred to a proper ship. Had it not been for her steadfastness of purpose, her extraordinary administrative ability, her patience and tremendous breadth of vision, as well as control of the London Times Fund, her purpose could never have been accomplished.

The Nightingale party arrived at Scutari with a group of 38 women on the 3rd of November 1854 and proceeded at once to the barrack hospital. This was a huge, old Turkish barracks, part of which had once been a stable, which was located over a cellar in part and over large, ineffective cesspools and sewers. Its use as a hospital had never been contemplated. Water and sanitary facilities were both at a premium, there was no heat and the building leaked. This small unwanted group arrived on a blustering winter day and were assigned to minute quarters only to find that the whole system of supply, medical care, and hospital services in the British army had completely broken down. The Battle of Balaklava had occurred ten days previously and the wounded and ill were arriving. The Battle of Inkerman was to take place the next day, and the debacle before Sebastopol that winter was about to occur. There were no food, clothes, blankets or hot water. Wisely Miss Nightingale forbade her nurses to make a move or intrude themselves until a request came from the doctors and from the start issued instructions that nothing was ever to be done unless requested by or under the supervision of the medical staff. This was the first time that any nursing service had ever been introduced into the British or any other army and it had to overcome hostility and suspicion on all sides. The first break came when the nurses were asked to help with the cooking. This had been almost non-existent before, but little by little a good general kitchen and various diet kitchens were established. A laundry was fitted up with sufficient water supply and stoves so that hot water was provided and the bedding and clothes could be washed, which until her arrival had not been done. The ill and wounded as they arrived from across the Black Sea were in frightful condition. Many had been on the ships from days to two or three weeks and were the victims not only of injury but of starvation and freezing. As they arrived in the already overcrowded hospital, in spite of the care and the increasing food supply that had become available through the efforts of Miss Nightingale, they were now subject also to hospital fever, dysentery

and cholera caused by the unbelievably bad sanitary conditions.

As the winter wore on the hospital mortality increased and it was noted that the mortality from one area in the hospital was almost 100 per cent. Reports going back to England stirred such a storm of protest that in February, 1855, a Sanitary Commission was sent to investigate the state of the buildings used as hospitals. As their work progressed, it was found that the sewers beneath the hospital were choked, overloaded and in places flooding back into the wards. In addition the water supply, which was totally insufficient, was also contaminated from these open privies and sewers. Finally, as the sewers were opened and cleaned and dead animals removed, conditions improved with corresponding prompt decrease in mortality. Had it not been for this commission, according to Miss Nightingale, the army would have been entirely destroyed, and as it was 73 per cent of eight regiments perished from disease in the winter of 1854 and 1855.

Notwithstanding the handicaps, the discouragements, the crowding of the hospitals, the seriously ill both from wounds and disease, the incredible long hours of work, Miss Nightingale kept detailed records and maintained a voluminous correspondence with England. On January 8, 1855 only two months after her arrival, she wrote: "I have written a plan for the systematic organization of these hospitals upon the principle of centralization under which the component parts might be worked in unison." In this she outlined a reorganization for various departments, the establishment of a corps of medical orderlies, changes in the cooking and food services, establishment of a medical school at Scutari, and spoke of the need for adequate medical statistics as to deaths and ages of those dying, types of treatment and all the data that have since become such an important part of our medical records. In another letter in the same month, she advised the abolition of the Post Purveyor and advised that each hospital should be an entity in itself. Her whole idea was less regulations and more service; in this way not only would the morale of the troops be improved and the men become self-respecting human beings, but lives would be saved.

Soon there was another long report on the internal reorganization of hospitals. This was forwarded on to Sir Sidney Herbert at the War Office, who had been responsible for sending Miss Nightingale to Scutari and with whom she had maintained a constant correspondence. In March 1855, Lord Panmure of the War Office dispatched Alexis

Soyer, chef at the Reform Club, who was ordered to take charge of the kitchens in the hospitals, which he did with immediate good effect and rapid improvement in the food service. As the fighting and disease quieted down in the spring, however, the jealousies among her own contingent and the hostility of Dr. Hall increased so that throughout the summer, although work had been accomplished as never before, the actual difficulties increased and it was not until the winter of 1856 that Miss Nightingale's position was finally clarified and published in a general order of the Army as in charge of all nursing in Turkey and the Crimea.

In addition to her work in the establishment of a nursing service and reorganization of hospitals over the opposition of Dr. Hall and many of the officers, she instituted a banking system for the British soldiers whereby they were enabled to remit money home. She established reading and recreation rooms and formed classes in the three R's so that a real reform was started in the British army, and the troops, instead of being left to their own devices with no outlets other than gin and women, under her guidance became a group of self-respecting men interested in and in correspondence with their homes and encouraged to save money and remit it to Britain. Herein we see the beginnings of a true information, recreation and education program for the enlisted soldiers, which was later to see its full development in the two World Wars.

Finally, with the end of the war and the last patient evacuated by the middle of July 1856, Miss Nightingale left for home on July 28 depressed and greatly broken in health. Both she and the British soldier emerged from the horrors of the Crimea as heroic figures. "Never again was the British soldier to be ranked as a drunken brute, the scum of the earth. He was now a symbol of courage, loyalty and endurance, not a disgrace but a source of pride." And, in turn, she had also established the nursing profession on a sound and humane footing as a group of women no longer dissolute and uneducated but with high principles in education and service.

Six months after her return home a confidential report to the War Office appeared as "Notes on Matters Affecting the Health, Efficiency and Hospital Administration of the British Army." She became obsessed with the need for reform not only in the medical services of the Army but in the War Office itself and worked steadily on her plans with Sir

Sidney Herbert, Dr. Sutherland and a few others who believed in her. In the course of her investigations she showed the mortality was several times as great in troop barracks in England as in the same localities in which they were situated. At last, after long struggle with the War Office, the Royal Sanitary Commission on the Health of the Army was appointed in the summer of 1857. In the plan prepared by her on the Health Administration for the Army, four subcommissions were recommended which were to begin to implement the recommendations of the plan:

1. Put the barracks in sanitary order
2. Found a statistical department of the Army
3. Institute an Army Medical School
4. Completely reconstruct the Army Medical Department, revise the hospital regulations and draw up a new warrant for the promotion of medical officers.

It was not until the end of the year, however, that these subcommissions were finally appointed with ample instructions. The struggle had really just begun and although she made great headway toward final victory in the next ten years with the reorganization of the Medical Department of the British Army, the opposition was too great against reorganization of the War Office itself, and although this plan lingered on until 1872, Miss Nightingale finally had to admit defeat. Fortunately before the death of Sidney Herbert, however, the Medical Department had been thoroughly reorganized and better hospitals and barracks provided in spite of the fact that Lord Panmure had his way in the building of an expensive, large, new hospital at Netley, the windows of which all faced to the northeast and which was quite without adequate sanitary provisions. All questions pertaining to the health and sanitary conditions in the British Army were referred to her, however, in new plans for administration.

She developed a cost accounting system for the Army Medical Service "which was put into operation between 1860 and 1865 and, 80 years later, was still in use. In 1947 the Selective Committee on Estimate reported favorably on it, commented that it worked admirably, so in other departments the systems installed within the last 20 years had been discarded."

In 1862 and 1863 she plunged into the midst of a discussion of venereal disease in the Army, vehemently opposed the continental system of licensing prostitutes and took the position that more attention

should be paid to instruction in the campaign against drunkenness and ignorance. In spite of her efforts, however, the House of Commons went against her and in 1864 passed the Contagious Diseases Act, which provided for police regulation and inspection.

In the spring of 1864 she supplied information for a speech by the Undersecretary of War who defended successfully the increased cost of hospital and medical services for the Army, which had risen from 19,000 pounds in 1853 to 295,000 pounds in 1864. A year later an attack on sanitary principles in the House of Lords met with no response as during the previous ten years the public had become educated in sanitation and the fruitful results of better medical care in the Army. Her main labors and interests were centered at home in the War Office and in her work on the Indian Sanitary Commission, and although inquiries were made of her from the United States as to her advice in the nursing situation in our own war between the States and she offered much information, still this was really a thing apart from her major interest. Nevertheless, as a result of her work in the Crimea and later in the reorganization of the Medical Department of the British Army, her labors there brought forth fruit here in the creation of our own Sanitary Commission and a complete revivification of the Army Medical Department. It may well have been through the inspiration of this reorganization that Surgeon General Hammond recommended similar reforms in our own medical department, such as the establishment of a medical school, a museum, the medical library, and the recording and accumulation of accurate statistics. Her struggles and final victory and emergence from the mires of the Crimean War and with the War Office in the Medical Department reorganization, may be well described by the majestic cadences in the introduction of Trevelyan's History of England:

> "Horns are heard blowing in the mist, and a confused uproar of savage tumult and outrage. We catch glimpses of giant figures—mostly warriors at strife. But there are ploughmen, too, it seems, breaking the primeval clod, and we hear the sound of forests crashing to the axe. Around all is the lap of waves and the cry of seamen beaching their ships."

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THE TWENTY-NINTH

ANNUAL GRADUATE FORTNIGHT

THE NEW YORK ACADEMY OF MEDICINE

October 15-26, 1956

SELECTED SUBJECTS OF CURRENT SIGNIFICANCE

MONDAY, OCTOBER 15

Evening Lectures

General Topic: THE PLACE OF HYPOPHYSECTOMY AND ADRENALECTOMY IN THE MANAGEMENT OF NEOPLASTIC AND METABOLIC DISEASES

1. Hypophysectomy in Man
   Rolf Luft, Karolinska Institute, Stockholm, Sweden
2. The Place of Hypophysectomy and Adrenalectomy in the Complications of Diabetes
   Laurance W. Kinsell, Institute for Metabolic Research, Oakland, California
3. The Place of Adrenalectomy in the Management of Severe Hypertension
   Charles C. Wolferth, University of Pennsylvania, Philadelphia, Pa.

TUESDAY, OCTOBER 16

Afternoon Panel Meeting

The Management of Disseminated Neoplastic Diseases

Moderator: David A. Karnofsky

Memorial Center for Cancer and Allied Diseases, New York City

WEDNESDAY, OCTOBER 17

Evening Lectures

General Topic: NEW DRUG THERAPY IN PSYCHIATRY AND HYPERTENSION

1. The Nature of Drugs with Mental Actions and Their Relation to Cerebral Function
   Harry H. Pennes, Eastern Pennsylvania Psychiatric Institute, Philadelphia, Pa.
2. New Drug Therapy in Psychiatry: Clinical Uses and Abuses in Mental Disorders
   Paul H. Hoch, Commissioner of Mental Hygiene, New York State
3. A Clinical Evaluation of Hypotensive Agents
   Irvine H. Page, Cleveland Clinic, Cleveland, Ohio

THURSDAY, OCTOBER 18

Afternoon Clinical-Pathological Conference

Hypertension

Moderator: Alfred Angrist

Albert Einstein College of Medicine, New York City

FRIDAY, OCTOBER 19

Evening Lectures

General Topic: AN EVALUATION OF PRESENT STATUS OF CARDIAC SURGERY

1. Physiological Considerations and Clinical Indications for Cardiac Surgery
   H. B. Burchell, Mayo Clinic, Rochester, Minn.
2. Surgery of Congenital Heart Disease
   Robert E. Gross, Children's Hospital, Boston, Mass.
3. Surgery of Acquired Heart Disease
   Frank Glenn, The New York Hospital-Cornell Medical Center, New York City

MONDAY, OCTOBER 22

Afternoon Panel Meeting

Surgical Management of Cardiovascular Disease

Moderator: Ludwig W. Eichna

New York University-Bellevue Medical Center, New York City

TUESDAY, OCTOBER 23

Evening Lectures

General Topic: THE ADRENAL CORTICAL STEROIDS AND THEIR ANALOGUES

1. Biosynthesis of Adrenal Cortical Hormones
   Gregory Pincus, The Worcester Foundation for Experimental Biology, Shrewsbury, Mass.
2. The Role of the Adrenal Cortical Steroids and Their Analogues on Electrolyte and Fluid Metabolism
   Louis J. Soffer, The Mount Sinai Hospital, New York City
3. Clinical Uses and Hazards of the Adrenal Hormones and Their Analogues in the Management of Rheumatic Diseases
   Joseph J. Bunim, National Institute of Arthritis and Metabolic Diseases, Bethesda, Maryland

WEDNESDAY, OCTOBER 24

Afternoon Clinical-Pathological Conference

An Endocrine Problem

Moderator: John G. Kidd

The New York Hospital-Cornell Medical Center, New York City

THURSDAY, OCTOBER 25

Evening Lectures

General Topic: POLIOMYELITIS

1. The Nature of Viruses
   Mark H. Adams, New York University-Bellevue Medical Center, New York City
2. Present Status of Attenuated Live Virus Poliomyelitis Vaccine
   Albert B. Sabin, The Children's Hospital Research Foundation, Cincinnati, Ohio
3. Recent Experience and Present Status of Killed Virus Poliomyelitis Vaccine
   Joseph E. Smadel, National Institutes of Health, Bethesda, Maryland

FRIDAY, OCTOBER 26

Afternoon Panel Meeting

Therapy of Infections

Moderator: David E. Rogers

Division of Infectious Diseases, The New York Hospital-Cornell Medical Center, New York City

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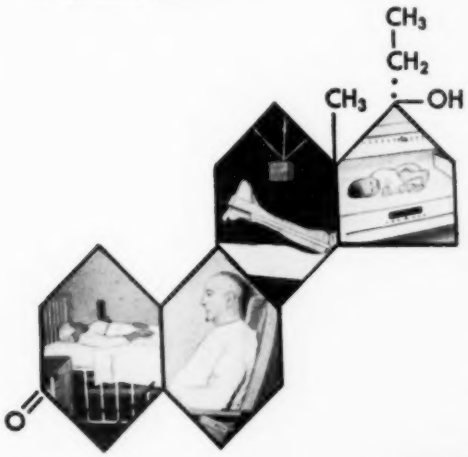

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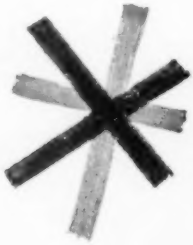

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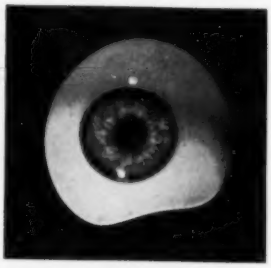

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References: 1. South. M. J. 31:233 (March) 1938. 2. Bull. New York Acad. Med. 19:478, (July) 1943. 3. Am. Heart J. 18:425 (Oct.) 1939. 4. Minnesota Med. 33:1102 (Nov.) 1950.

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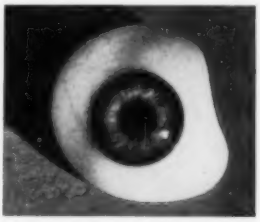

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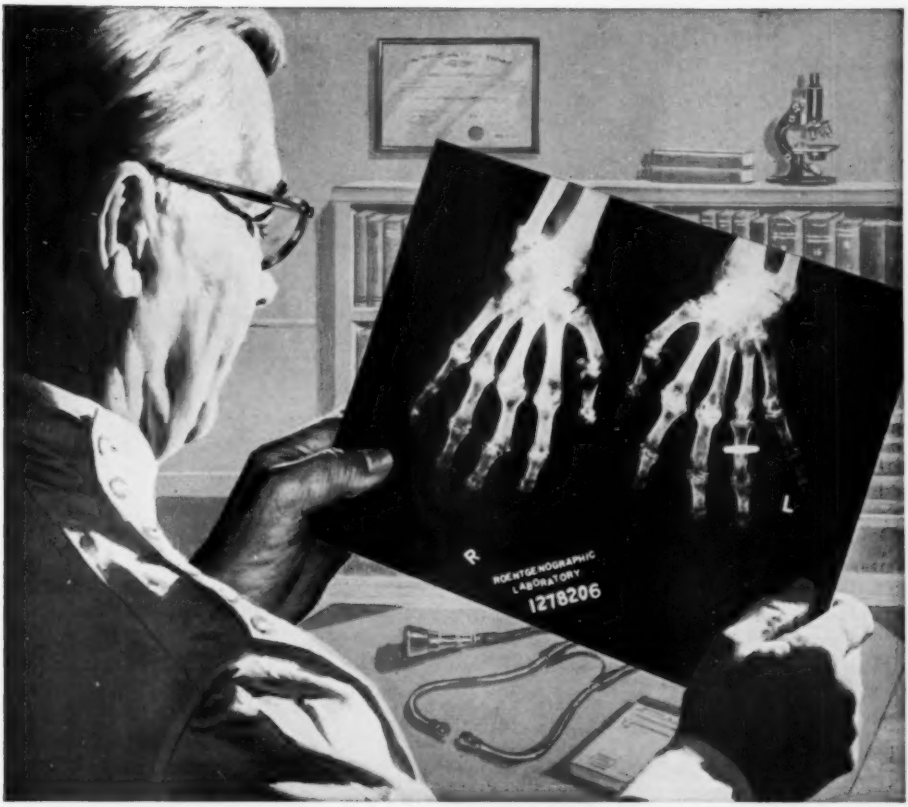

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REFERENCES:
1. J.A.M.A. 159:645 (Oct. 15) 1955.
2. J.A.M.A. 158:386 (June 4) 1955.

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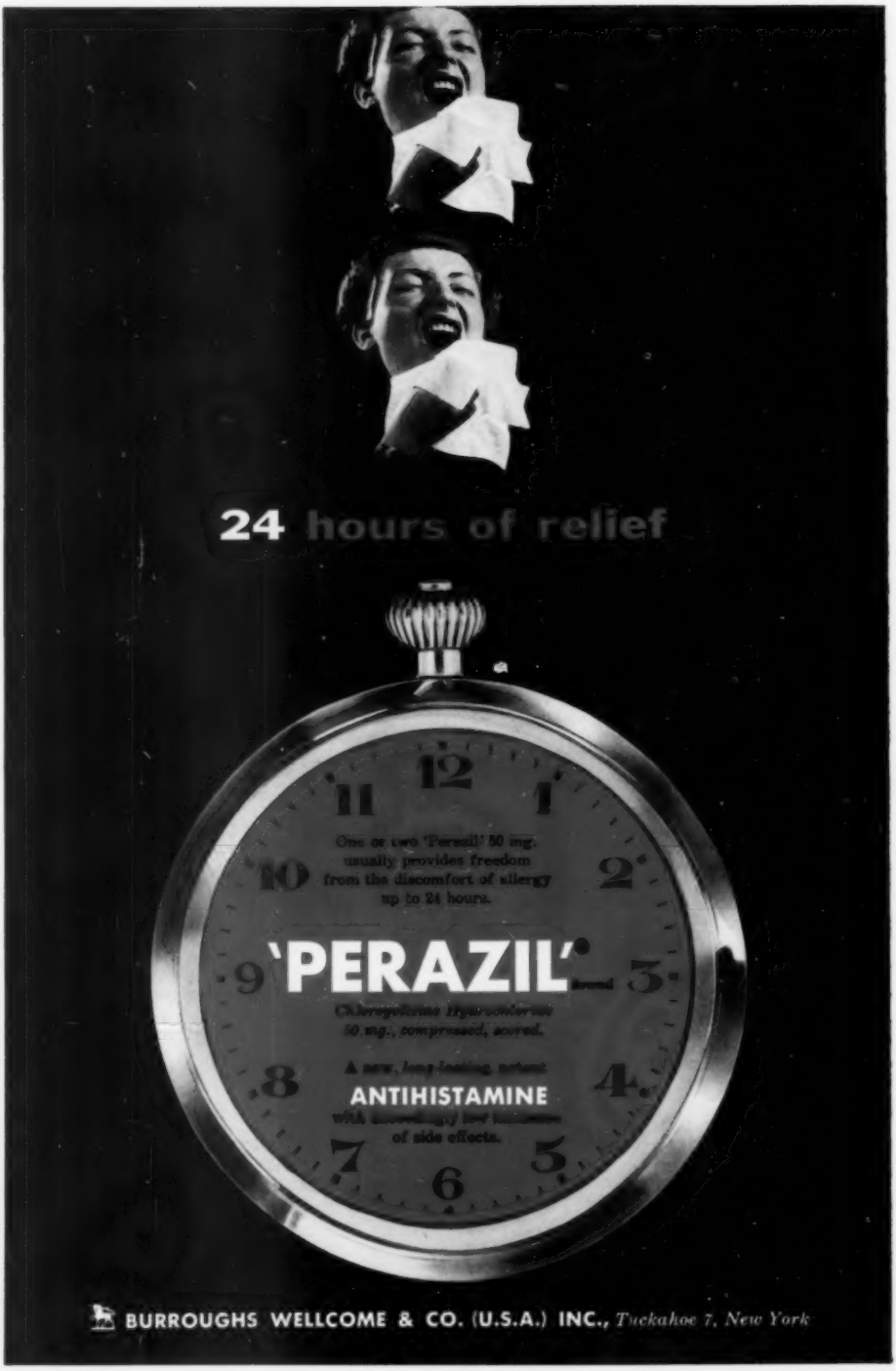
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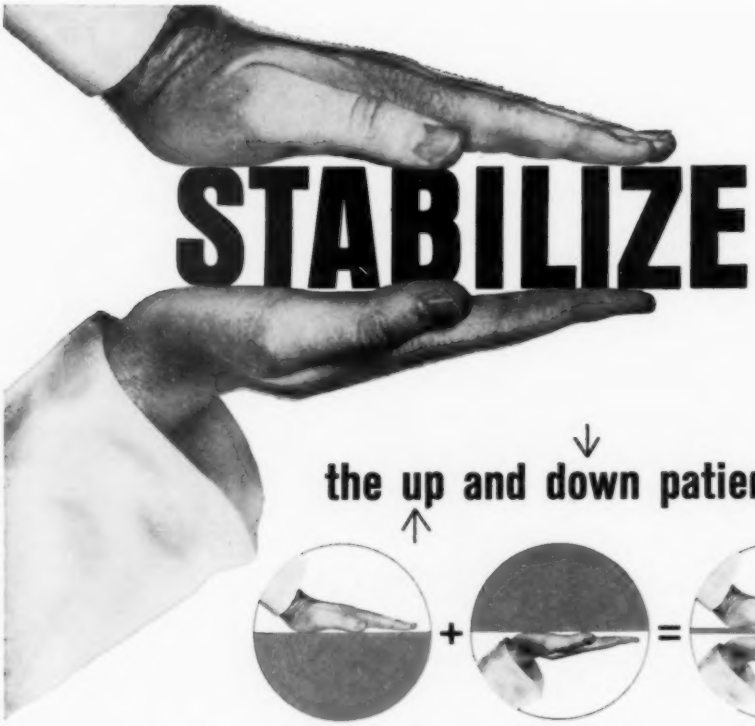

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1. Arnoff, B.: Personal communication. 2. Lazarte, J. A., and Petersen, M. C.: Personal communication.

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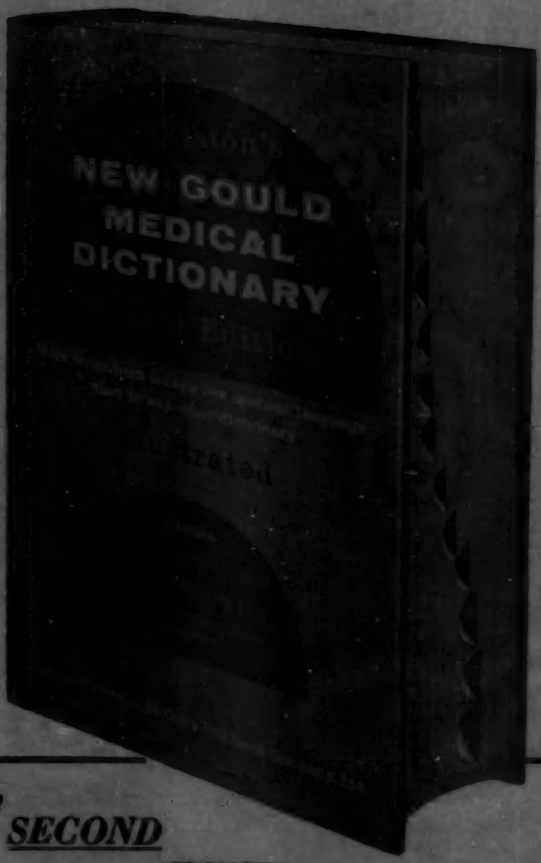

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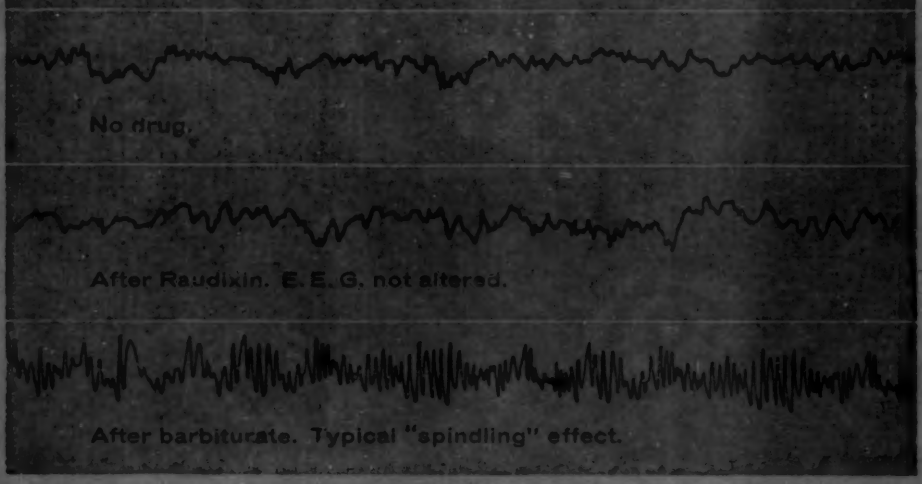

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